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RECENT STUDIES ON SPIROCHETES IN GENERAL PARALYSIS *

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During the last five years a number of important articles dealing with the location of spirochetes in general paralysis have appeared, especially in the German literature, and I wish to try to summarize briefly the main facts, comments and theories that have resulted from the studies of three of the best investigators, namely, Jahnel,[1] Sioli [2] and Hauptmann.[3]

Jahnel [4] of Frankfurt has devised an excellent silver stain for spirochetes in the central nervous system. This stain brings out the organisms with great sharpness on a yellow, or yellowish-brown background; it usually leaves unstained those elements of nervous tissue (axis cylinders, etc.) that have heretofore been so troublesome to all who have tried to study spirochetes. It is largely due to the excellence of this stain that others, besides Jahnel himself, have been stimulated of late years to study the position, the distribution and the activity of these parasites in the brain and spinal cord.

SPIROCHETES IN GENERAL PARALYSIS; PARTS OF BRAIN MOST LIKELY TO SHOW THEM

By Jahnel's method spirochetes have been found in from 25 to 50 per cent. of the cases studied. Still higher percentages have been obtained by brain puncture during life and dark field illumination

* Read at interhospital meetings held at Hudson River State Hospital and Utica State Hospital, March, 1922.

1. Jahnel, F.: Ueber einige Beziehungen der Spirochäten zu dem paralytischen Krankheitsvorgang, Ztschr. f. d. ges. Neurol. u. Psychiat., Orig. **42**:21-88, 1918. Die Spirochäten im Zentralnervensystem bei der Paralyse, ibid., Orig. **73**:310-335, 1921. Das Problem der progressiven Paralyse, ibid., Orig. **76**:166-182, 1922.

2. Sioli, F.: Die Spirochaete pallida bei der progressiven Paralyse, Arch. f. Psychiat. **60**:401-464, 1919.

3. Hauptmann: Spirochäten und Hirnrindengefässe bei Paralyse, Ztschr. f. d. ges. Neurol. u. Psychiat., Orig. **57**:122-173, 1920. Klinik und Pathogenese der Paralyse im Lichte der Spirochätenforschung, ibid., Orig. **70**:254-299, 1921.

4. Jahnel's method is described by Stevenson, George S.: Two Recent Improvements in the Staining of Spirochetes in Nervous Tissue, Arch. Neurol. & Psychiat. **7**:349 (March) 1922.

(Valente, 70 per cent.). Spirochetes are believed to be present in all cases of general paralysis, and the longer one hunts and the greater the number of areas studied, the smaller is the percentage of failure to find them.

Spirochetes may be found in any region of the brain, but all of the authors agree that the frontal lobes, especially the anterior parts of these where the changes in the tissues are greatest, offer the best opportunities for finding them. They are seldom seen in the pia mater; seldom in the outer layer of the cortex; almost never in the white matter. As a rule, they are most abundant in the middle and deeper

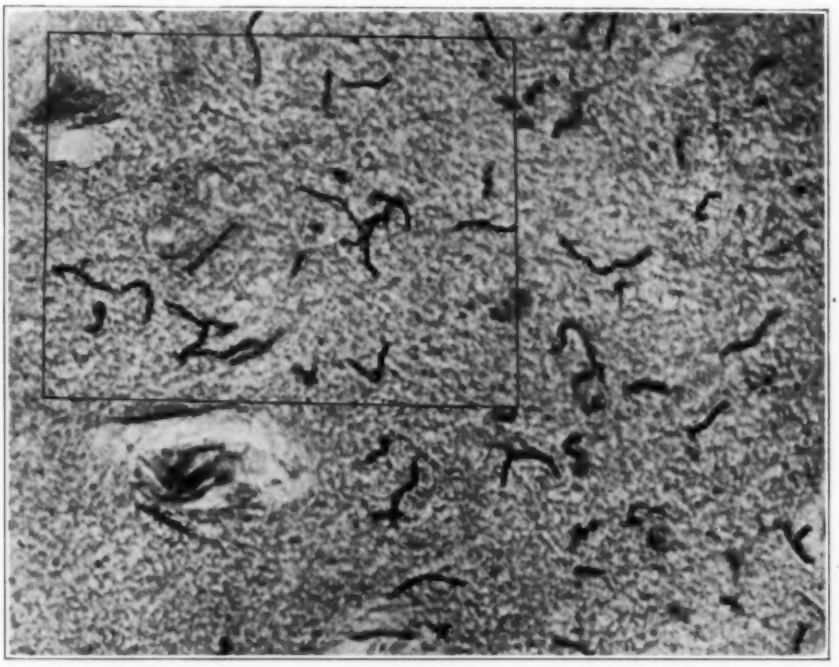

Fig. 1.—Photomicrograph of spirochetes in diffuse distribution; 4 mm. Zeiss objective, ocular No. 6. This field is from a part of a section in which spirochetes were abundant; in many other parts of this section none were found.

This illustration and Figure 2 show spirochetes from the left frontal region of a patient with general paralysis, C. D., of the Institute collection; received from Willard State Hospital in 1911. The brain when received showed evidence of partial decomposition; it had been kept in 10 per cent. liquor formaldehydi for about ten years before the sections were removed. They were stained by Dr. G. S. Stevenson by Jahnel's method.

layers of the gray matter, a point of great practical importance in searching for them. They are rather more likely to be found in the gray matter along the fissures than at the tops of the convolutions and

may (rarely) when stained be so numerous as to cause dark spots visible either to the naked eye or on low magnification.

NUMBER OF SPIROCHETES FOUND IN DIFFERENT CASES

In most cases the number found is decidedly small, but occasionally they are found in swarms or colonies in almost uncountable numbers.

CASES MOST FAVORABLE FOR FINDING SPIROCHETES

Spirochetes are most readily found in cases that run a stormy course, with convulsions, sudden paralytic attacks or acute seizures of various

Fig. 2.—Enlargement of that part of Figure 1 which is included in the square. The parasites are not all of the same size. Zeiss one-twelfth oil immersion; projection ocular No. 4.

kinds. If a patient dies immediately after a seizure, the chances of finding spirochetes are greatest, for they seem to be quite fleeting and to increase and to decrease rapidly in successive waves, so that if death is delayed the hypothetical wave may have subsided.

They are by no means regularly more numerous where the cellular exudates are greatest; in fact, the spirochetes often (perhaps most often) lie in places in which the exudate is slight. Here they seem to exist with no special reaction on the part of the tissues to their presence. This fact has led to the hypothesis that it is only when they disintegrate

that they light up a reactive process with abundant exudate and destruction of the tissue.

The histologic changes in the brain tissue must not be attributed to the spirochetes seen with the microscope at the time of death, for these spirochetes form only a momentary picture caught at the time of dissolution of the host; the tissue changes are rather the sum of all the processes that have been going on for months or years, and are not a result of the fleeting spirochete picture seen at the particular time when death arrives. The actual spirochete picture at the time of death is probably different from the one seen at the time of examination

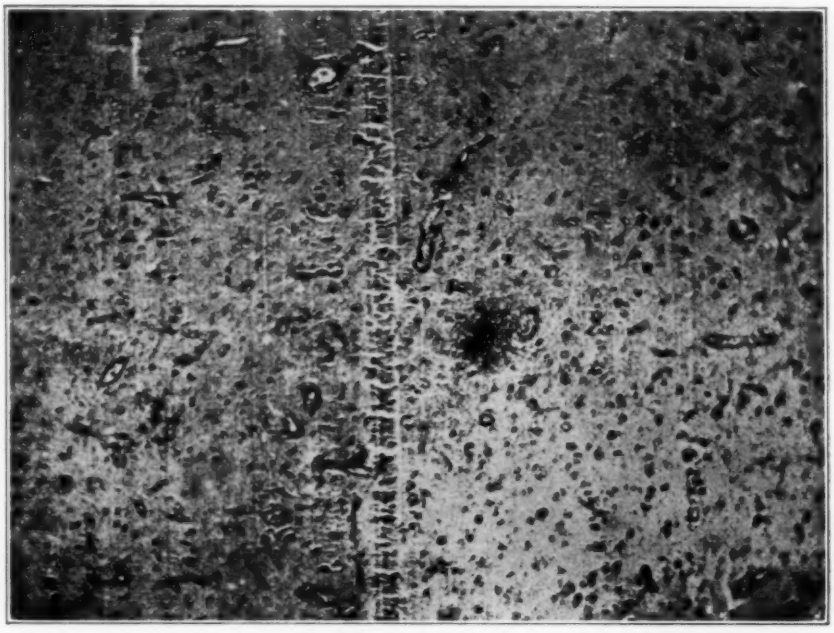

Fig. 3.—General paralysis: This shows, in low magnification, a swarm-like colony of spirochetes just to the right of the center of the figure.

Figures 3 and 4 are photographic reproductions from Plate 4, Figures 15 and 16, in F. Jahnel's article "Ueber einige Beziehungen der Spirochäten zu dem paralytischen Krankheitsvorgang," Ztschr. f. d. ges. Neurol. u. Psychiat.; Orig. **42**:21, 1918. Jahnel's method of staining.

because of postmortem migration of the parasites, possibly into parts that they may not be able to enter during the life of the patient; the blood vessels, for example.

VIABILITY AND RESISTANCE TO DECOMPOSITION

However rapidly spirochetes may increase and diminish during the life of the patient, after the patient is dead they may continue to live

for hours or days. Hauptmann found them actively motile in a case of general paralysis forty-eight hours after death, so that in a reasonably early necropsy one would expect to find their viability little affected, and they would be suitable for inoculation experiments in animals. Owing to their active motion, they are believed to change their position in the brain rapidly after death, as already stated, so that necropsy should be made as quickly after death as possible, and the brain should be fixed at once, if anything resembling true relations is to be preserved. In extremely rare instances some of the organisms, as shown in Figure 14, are believed to have wandered through

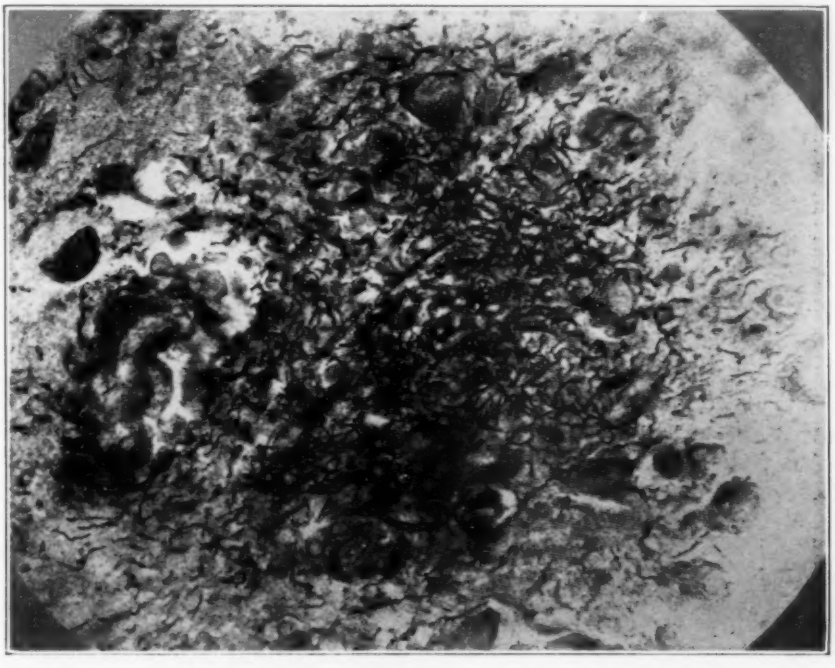

Fig. 4.—The same colony as in Figure 1, more highly magnified. Dr. Jahnel states that such colonies are sometimes visible to the unaided eye.

the walls of the blood vessels, after the death of the patient, and in this way to have entered the blood stream where (I repeat) they have never been demonstrated antemortem with the microscope, although a few of the many experiments with animals have apparently shown that spirochetes may at times exist in the blood of patients with general paralysis.

Spirochetes are strongly resistant to decomposition, and the dead organisms have been demonstrated in the brain about thirty days after decomposition had been allowed to go on.

FORM OF SPIROCHETES IN GENERAL PARALYSIS AND IN SYPHILIS

No difference in form, shape or size has been established between the spirochetes of general paralysis and those found in ordinary syphilis. Noguchi described, in general paralysis, at least three morphologic varieties—small, medium and large—and the same forms are found in ordinary syphilis. Later we shall consider some biologic peculiarities in the two groups of spirochetes, but the individual organisms in each group are alike in shape and in staining reactions.

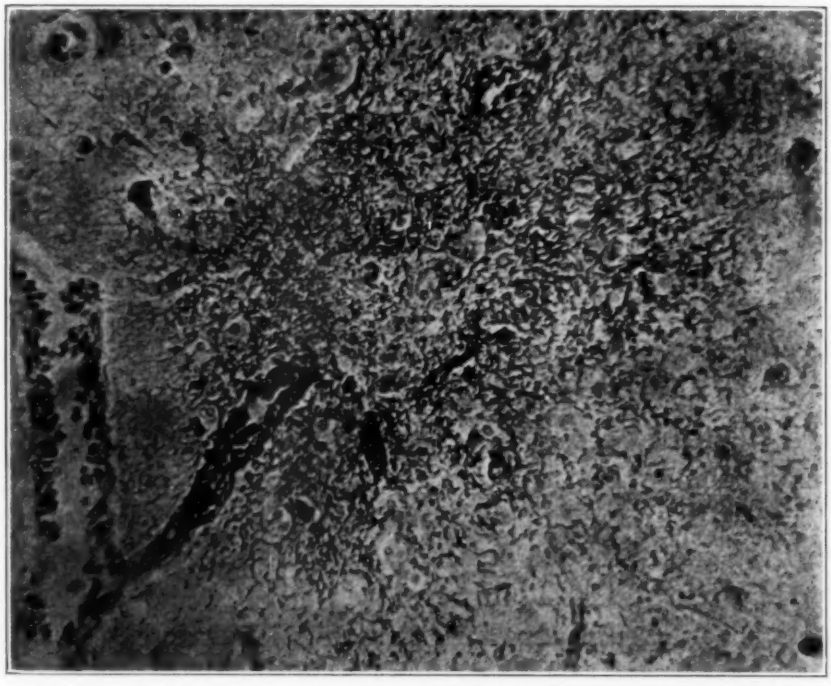

Fig. 5.—A localized collection of spirochetes which are not only within the nervous tissues, but are also within the walls of the blood vessel. This is to be interpreted, according to Hauptmann, as a proliferation of spirochetes in tissues in which the blood vessels do not offer enough resistance to prevent the parasites from invading their walls.

Figures 5 and 6 are photographic reproductions from Figures 6 and 7 in Professor Hauptmann's article, "Spirochäten und Hirnrindengefässe bei Paralyse," Ztschr. f. d. ges. Neurol. u. Psychiat., Orig. **57**:122, 1920.

PECULIARITIES IN GROUPING AND DISTRIBUTION OF SPIROCHETES IN GENERAL PARALYSIS

Three main types of distribution have been described:

1. A diffuse type, in which the organisms are diffusely scattered, without order, usually in small numbers, rarely in large numbers, throughout the gray matter of the nervous tissues.

2. A focal type in which they are grouped together, sometimes in swarms like swarms of bees, often in immense numbers, but with no relation to blood vessels or any other elements in the nervous tissues. These swarm-foci are not common.

3. A vascular type, still rarer, in which they are especially concentrated about the blood vessels, the walls of which they often penetrate. This vascular type is believed by Jahnel to be only a subvariety of the focal or swarm type, for the reason that the vascular type

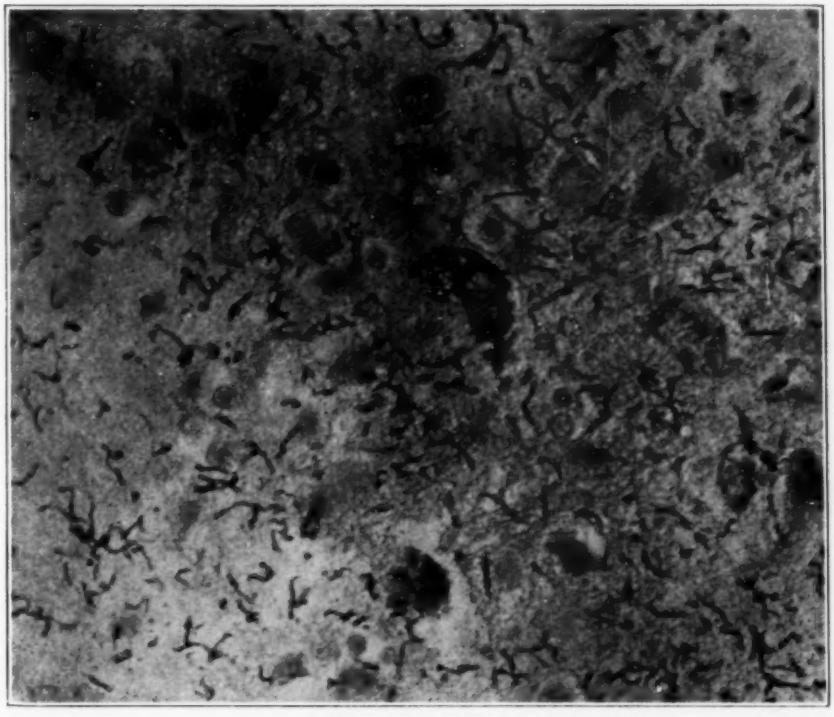

Fig. 6.—Diffuse distribution of spirochetes in combination with blood vessels which contain the organisms; no genetic connection exists between the spirochetes in the vessels and those in the tissues.

occurs in rather sharply bounded foci, and the spirochetes do not stream out along the blood vessels beyond the limits of the focus, nor do they follow the branches of the vessels as they would do if they belonged strictly to the vessels or were derived from them.

Hauptmann has especially studied this vascular form, and believes that the spirochetes here do not, generally speaking, come out of the blood stream to enter the parenchyma, but that they go in the reverse direction, from the parenchyma toward the vessels, and penetrate the latter; how much of this penetration may occur postmortem is not known.

It is in these vascular foci, and here only, that a spirochete has rarely been found in the lumen of a blood vessel among the corpuscles.. They have never been seen in the blood stream outside of such foci. None of the authors cited believe that these swarms or focal collections have been directly derived from the blood stream, though this possibility may be left open. They think the best explanation for these masses is that the spirochetes have proliferated in loco, an explanation that seems to be justified by the phenomena observed. It seems to be pretty generally agreed, as previously stated, that the organisms may change their rela-

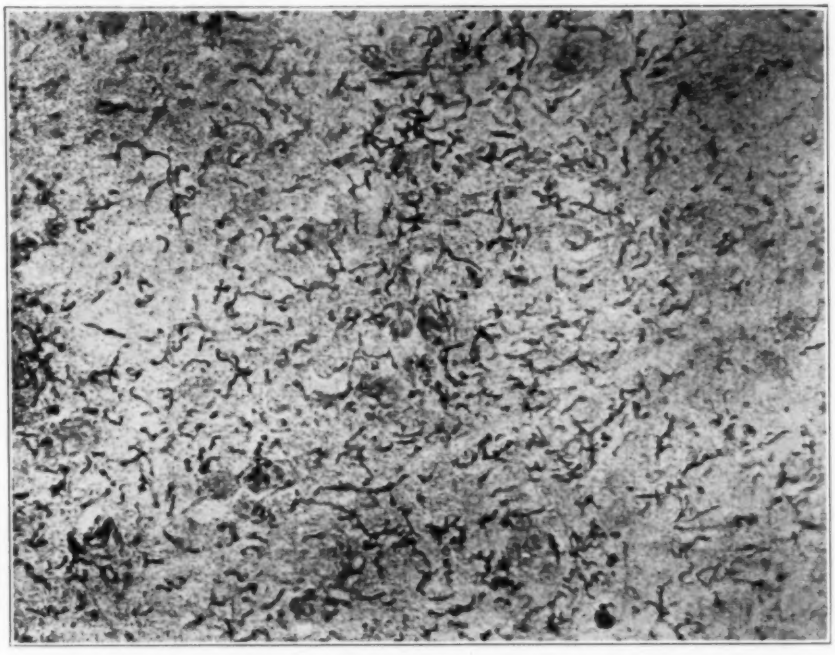

Fig. 7.—Diffuse distribution of spirochetes with no relation to blood vessels.

Figures 7 and 8 are photographic reproductions from Figures 12 and 13 in Professor Hauptmann's article.

tions and position rapidly, receding in one part of the brain and flaring up in another.

RELATION OF SPIROCHETES TO NERVE CELLS, NEUROGLIA CELLS AND EXUDATE

No one has ever satisfactorily demonstrated that spirochetes exist within nerve cells, although they may be grouped around nerve cells. It is questionable whether they have been seen in neuroglia cells. They have sometimes been found among the cells of the exudate, but have not been conclusively shown to lie within the cells themselves.

METHOD OF INCREASE OF SPIROCHETES IN GENERAL PARALYSIS AND PATHWAYS BY WHICH THEY SPREAD

Little is known concerning the method of increase of spirochetes in general paralysis. We know practically nothing of the rapidity of progression of spirochetes and the paths by which they travel. It is considered highly probable that they proliferate and spread locally in the gray matter, but it is not likely that they are spread from place to place in the brain to any great extent by the blood stream, or by the cerebrospinal fluid, for, with the exceptions previously mentioned,

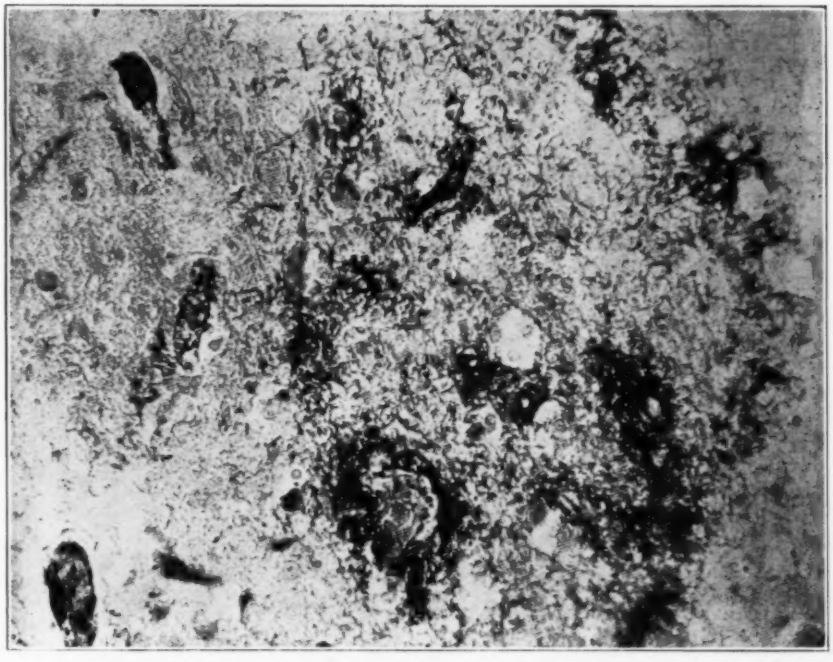

Fig. 8.—Localized massive girdling of the walls of blood vessels (Wallbildung) by spirochetes and invasion of the walls of the vessels by the parasites.

where postmortem migration is probable, they have never been seen with the microscope in general paralysis in blood, cerebrospinal fluid or in the centrifugalized sediment of the fluid.

We must remember, however, that in some instances animals have been reported as infected by injections of blood or of spinal fluid from patients with general paralysis, though many investigators have always failed to obtain results by such injections. Jahnel himself thought he succeeded with one rabbit after seven months of incubation, but he could not keep the strain to study its characteristics. Valente failed utterly in 103 animals, using puncture material from brains of living

patients with general paralysis, also the blood and spinal fluid. Forster and Thomasczewski likewise failed in sixty rabbits, with brain puncture material obtained from fifty-three living patients with general paralysis.

If the apparently successful injections can be trusted, we must assume that, at least sometimes, spirochetes are present in the blood and spinal fluid in general paralysis, but whether the blood and fluid play an essential rôle in spreading them is still doubtful. The undecided question always rises in these cases as to whether the ordinary lesions of somatic syphilis coexist with the specific lesions of general paralysis.

Fig. 9.—Spirochetes in the vessels and in the nervous tissue.

Figures 9 and 10 are photographic reproductions from Plate 3, Figures 11 and 12, in Jahnel's article.

RELATION OF SPIROCHETES IN GENERAL PARALYSIS TO WASSERMANN REACTION

Sioli reports one case of general paralysis which showed spirochetes in the brain although the Wassermann reaction (twice repeated) was negative in both blood and spinal fluid. Valente, a Portuguese investigator, reports three cases with positive spirochete findings but negative Wassermann reactions in the spinal fluid. In my experience I can recall no case of general paralysis, confirmed by the microscope, in which the fluid Wassermann test, if made, was consistently negative. Jahnel regards a positive Wassermann reaction as a probable index of the presence of spirochetes in the body. I think we should look on the

negative Wassermann reactions (not further described especially as to treatment) with some skepticism, as defects in technic or reagents are not rare; the personal equation of the serologist has also to be considered, and it is possible to mix up specimens.

HOW DO SPIROCHETES REACH AND ENTER THE BRAIN IN GENERAL PARALYSIS?

This is still an open question. Some authors, such as Valente, think that spirochetes may enter the central nervous system at the time of

Fig. 10.—General view showing the position of spirochetes in both nervous parenchyma and in blood vessels. The dark masses are due to aggregations of spirochetes in large numbers.

the early syphilitic meningitis, when the whole system is flooded with spirochetes; at this time, under unknown conditions, they "break through the neuroglial boundary membrane." Such views do not seem to account satisfactorily for the long incubation period of ten to twenty years in general paralysis. That spirochetes do enter originally by the blood or meningeal route, however, seems most probable.

LONG INCUBATION PERIOD AND EHRLICH'S VIEWS OF RECIDIVE STRAINS

The neurorecidive theory of Paul Ehrlich does take the incubation period into account, and while admittedly only a theory which is unten-

able at present on the basis of established facts, it is extremely suggestive, although it makes no attempt to show just how the spirochete of general paralysis actually enters the nervous tissue. According to Ehrlich, spirochetes enter the body, including the meninges, at the time of infection, and at first they increase without limit. After a time the body produces a protective material (or antibody) which kills off most of the micro-organisms; but some are more resistant, remain alive and produce a new generation of spirochetes (recidive strain No. 1). Against this new generation of spirochetes the body again reacts by antibody formation; this same cycle is repeated again and

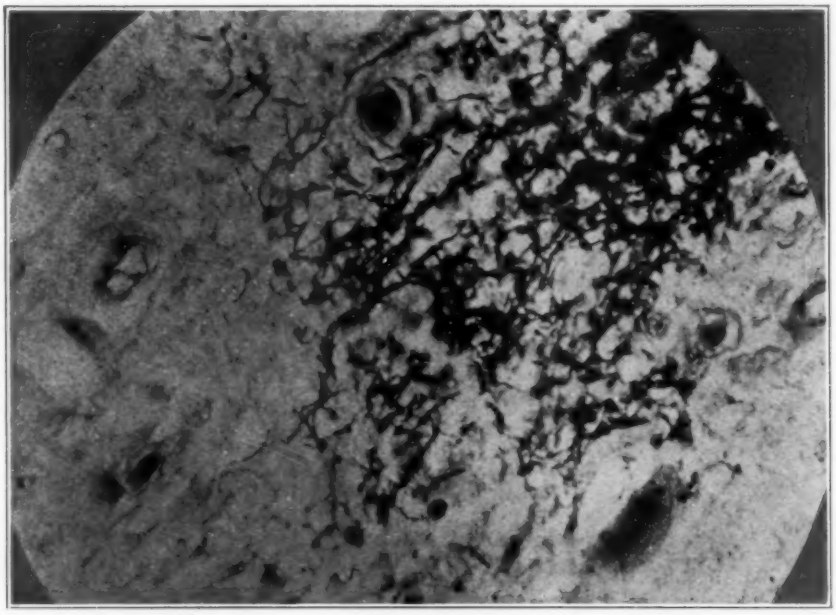

Fig. 11.—Spirochetes in swarmlike colonies. Note the few spirochetes outside of the colony itself.

Figures 11 and 12 are photographic reproductions from Plate 4, Figures 9 and 12, in an article by Dr. F. Sioli, entitled "Die Spirochaete pallida bei der progressiven Paralyse," Arch. f. Psychiat. **60**:401, 1919.

again, always with the formation of higher and higher recidive strains of spirochetes. Such spirochetes as survive in this recidive process (if I understand Ehrlich) would belong to the original syphilitic strain that entered the body at the time of infection, but would be altered biologically—a special selection of those best fitted to survive; that is, a straight syphilitic strain, but modified by the conditions of growth within the host. Moreover, as the recidive strains progress, the places in which the syphilitic organisms can be found grow fewer and fewer;

the parasites seem to retire in the later stages of syphilis to certain circumscribed foci, the central nervous system being possibly one of these foci. It is only in the secondary stages and early stages of syphilis that one finds a general spread of spirochetes and generalized lesions. In the tertiary stages of syphilis the lesions are much fewer in number and more focal in position.

Thus the recidive theory of Ehrlich accounts for an indefinite period of time during which the spirochetes are supposedly undergoing biologic alterations, and are receding more and more from general distribution

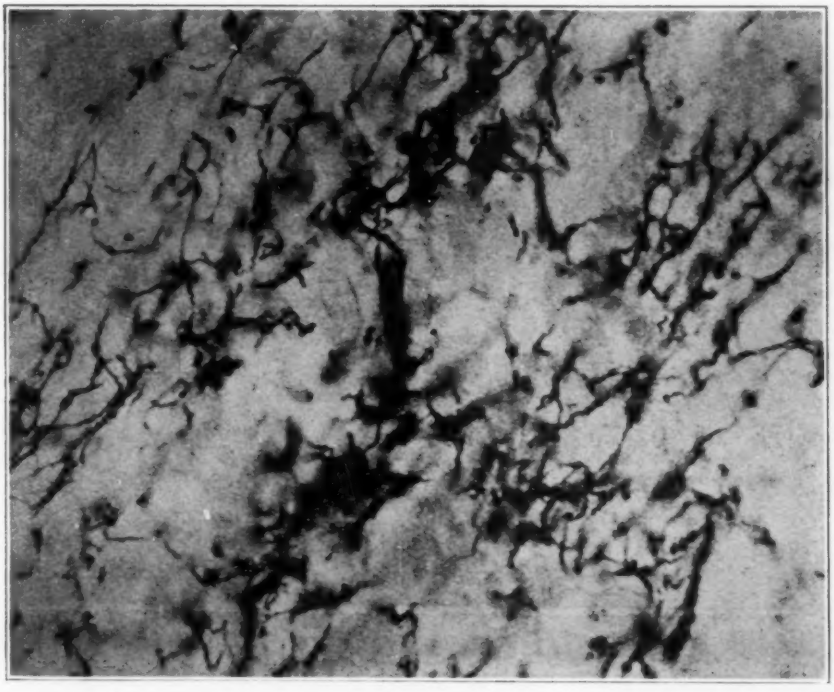

Fig. 12.—A similar swarmlike colony of spirochetes more highly magnified.

to more circumscribed locations, as is the case in tertiary, or late syphilis, and in general paralysis.

This is hardly the place to discuss the many other views and theories dealing with the latent period of syphilis and the habitat of the organisms between the time of primary infection and the outbreak of the symptoms of general paralysis. This subject has been dealt with by Jahnel [5] in a later article.

5. Jahnel: Das Problem der progressiven Paralyse, Ztschr. f. d. ges. Neurol. u. Psychiat. **76**:166-182, 1922.

As long as spirochetes are in the general mesoblastic tissue, they are usually regarded as relatively accessible to treatment. Those that reach the parenchyma of the brain appear to be extraordinarily inaccessible, as even the most diffusible drugs are reported scarcely to enter the brain substance at all, and arsenic, so far as I know, has not been demonstrated in the parenchyma of the brain after treatment with arsenical compounds.

It is recognized that histologic changes in the brain of patients with general paralysis may be advanced, although clinical symptoms are recent. Alzheimer, in patients who had previously had syphilis, not

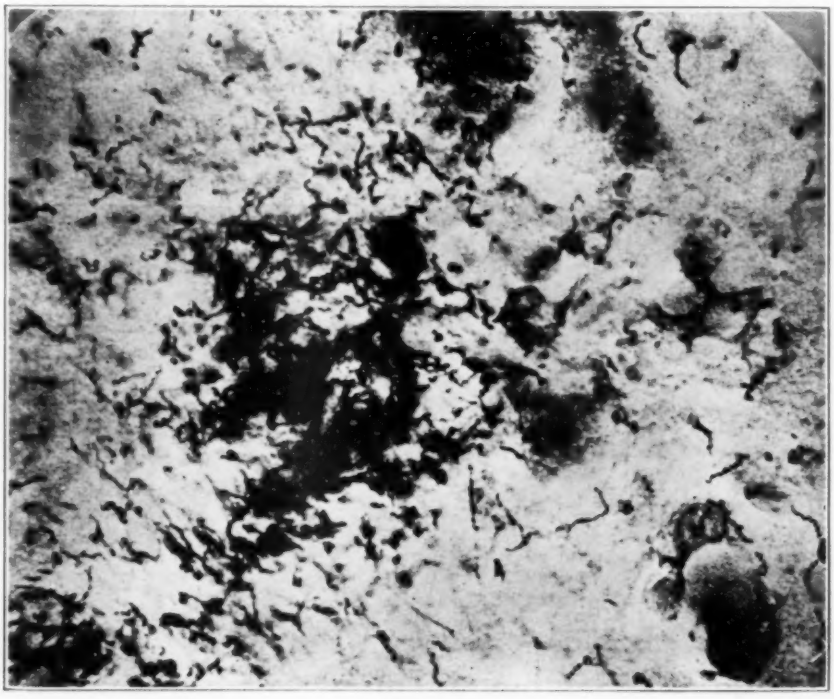

Fig. 13.—Enormous numbers of spirochetes in the blood vessel walls and in the neighboring tissue.

Figures 13 and 14 are photographic reproductions from Plate 7, Figures 1 and 5, in the article by Dr. F. Sioli.

infrequently found considerable collections of lymphocytes and plasma cells in the meninges, without evidence of cortical disease in the brain. It seems highly probable that the spirochetes reach the meninges early in the infection in a large proportion of cases of syphilis, but in most cases they do not get a foothold in the parenchyma. They have been shown, with the microscope, by various authors to exist in the spinal fluid in patients with early syphilis—not in those with general paralysis.

Again in early syphilis, untreated, about 80 per cent. of the patients, according to Sioli, who bases his figures on an extensive material collected from the literature, have shown an abnormal state of the spinal fluid, either in the Wassermann reaction, cell count, albumin content or in some other manner. In the second stage of syphilis only about 33 per cent. of the patients showed similiar changes in the fluid; in the third stage and latent stage, about 23 per cent. So we can say that in the early stages of syphilis changes in the spinal fluid are at least frequent, but that later the changes spontaneously disappear in many cases and persist in others.

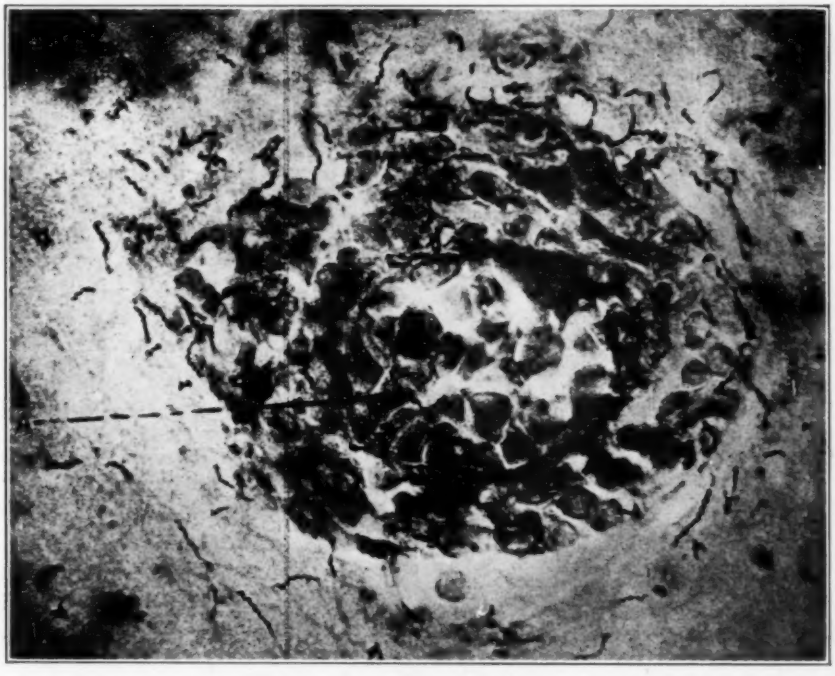

Fig. 14.—Spirochetes in the wall of a blood vessel, also in the zone of the infiltrate, and one (*A*) within the lumen. The latter parasite probably reached its position among the corpuscles after death.

IS GENERAL PARALYSIS CAUSED BY A SPECIAL STRAIN OF SPIROCHETE, A NEUROTROPIC STRAIN, DIFFERENT AT THE BEGINNING FROM THE SPIROCHETE OF ORDINARY SYPHILIS?

In favor of this view are groups of patients with general paralysis and tabes infected from the same source: cases of conjugal infection in which husband and wife both have general paralysis of tabes; infections among members of the same family who develop general paralysis or tabes.

To balance such facts is a report of group infection, likewise from a common source (Eichelberg, reported by Sioli), in which all of the infected members, thirteen in number, developed ordinary syphilis except two; one of these developed general paralysis, the other tabes.

Certain writers, especially Levaditi and Marie, favor the view that general paralysis is caused by a spirochete with special affinity for the nervous system—a neurotropic strain.

Levaditi and Marie inoculated the testes of rabbits with blood from patients with general paralysis. They obtained, in some cases, lesions and a strain of spirochetes which they transmitted to other rabbits; thus they obtained what they regarded as the true virus of general paralysis. They compared this general paralysis virus experimentally with a syphilitic strain of spirochetes that had been transmitted to rabbits about ten years previously from a syphilitic primary sore, and had been carried on from rabbit to rabbit ever since. They found marked differences in the two strains of spirochetes, as shown in the table.

Comparison of Dermotropic and Neurotropic Virus

Dermotropic Virus (Syphilitic)	Neurotropic Virus (General Paralysis)
Incubation: about 14 days	Longer, up to 127 days
Deep-seated infiltration	Superficial changes
Primary lesion lasted about 90 days	Slower, 169-195 days
Pathogenic for rabbits, lower apes, chimpanzee and man	Not pathogenic for apes and man, weakly so for rabbits
Immunity to reinfection with this syphilitic strain after recovery, but still susceptibility to the general paralysis strain	Immunity to the general paralysis strain after recovery, but still susceptibility to the syphilitic strain

Thus a so-called crossed immunity does not exist in animals. In man, however, patients with general paralysis are practically always immune to syphilitic strains, so the foregoing does not apply to man. The spirochete in general paralysis, however, differs from that of syphilis in being only slightly infectious, and incubation is long.

Jahnel says that as Levaditi and Marie, in their experiments, failed to exclude or even to mention an often-described rabbit disease in which spirochetes resembling those of syphilis are present, he cannot accept their results.

The authors we are summarizing agree that a "lues nervosa" is by no means proved. It is not even a necessary assumption; much more attention should be given to the soil, i. e., the individual in which the spirochete grows. Plaut thinks those who have metasyphilis may have from the beginning a special and peculiar reaction toward syphilis; that

is, endogenous factors may be at work. Plaut refers to the mild course and slight reactions to syphilis that some patients show who later develop general paralysis.

SPIROCHETES IN OTHER ORGANS THAN THE BRAIN IN GENERAL PARALYSIS

Until within the last year or so the following statement was considered to be justified: "There is not one well authenticated case of general paralysis in which spirochetes have been found in any organ except the brain" (Jahnel). Jahnel for years had searched, as he said, all possible organs extensively and never found them (one in the lung). Lately he has found them in the aorta. Of course even here a mixture of syphilis and general paralysis cannot be absolutely excluded.

The presence of spirochetes at times in the blood and spinal fluid in general paralysis, as shown by animal inoculations, has already been referred to, though the organisms have never been found with the microscope in either blood or fluid except as a possible postmortem migration into the blood, as already mentioned. They have been found in the meninges (Jahnel) in both general paralysis and tabes, rarely it is true, but sometimes in considerable numbers, so that one cannot say that in general paralysis spirochetes are strictly limited to the parenchyma of the brain. Neither can one say (with facts to support the statement) that in cerebral syphilis spirochetes are limited to the interstitial tissues, or mesoblastic tissues. In a few cases of cerebral syphilis in which adequate search for spirochetes has been made (e. g., Strassmann, Versé), occasionally some of the parasites were found in the parenchyma, though most were in the interstitial tissues. There is, however, almost nothing accurately known about the distribution of spirochetes in cerebral syphilis proper, and the question requires much more study.

General paralysis is, however, essentially, but not absolutely, a spirochetosis of the brain, and the terms "lues parenchymatosa" and "lues interstitialis" are not justified by present knowledge.

With the idea that there must be other retreats in the brain for spirochetes than those so far known, Jahnel searched extensively for a possible place where they could always be found, but without success. He found them in the pia mater of the cerebellum, in the cerebellar veins, in the pia mater of the pons and in the walls of the basilar artery. Immediately underneath a meningeal focus showing spirochetes the brain substance may or may not contain them, and an extensive focus of spirochetes may be present in the brain substance with clear meninges above this focus. He makes no attempt to decide whether they have spread from the meninges to the brain or vice versa.

He believes this meningeal spirochetosis, especially that in the cerebellum, is of great significance for the future, even though this significance is as yet unknown. This meningeal spirochetosis may form a bridge between general paralysis on the one hand and certain forms of cerebral syphilis on the other.

Some years ago I tried to bring out, in a paper read in Baltimore, that histologically there are sometimes all grades of transition between general paralysis and so-called cerebral syphilis, and that general paralysis is only a special form of cerebral syphilis. The foregoing findings of Jahnel, so far as they go, tend to confirm that view which was regarded by Mott at that time as erroneous.

SPIROCHETES IN TABES

In later work on tabes the organisms have been found by Jahnel in the pia-arachnoid and in the connective tissue sheaths of the posterior nerve roots. Jahnel included the dura mater in his sections, as otherwise much of the arachnoid which contains the spirochetes was lost. He warns against associating a chance collection of spirochetes in such places with anatomic lesions that may be present there: for what we see anatomically in these cases is the end result of processes that have lasted for years, and the same is true of general paralysis. Versé found many spirochetes in the posterior nerve roots of a case which clinically had neither tabes nor root symptoms, so the mere presence of spirochetes is not the only thing needed to produce tabes. A text on which Hauptmann writes an article is the following: "The spirochetes alone do not do it." Just as the presence of the tubercle bacillus, which most of us probably have harbored at times, is not the only condition for the production of tuberculosis, so the presence of *Spirochaeta pallida* is not the only condition needed in the production of general paralysis.

SUMMARY AND CONCLUSIONS

1. We have at last, thanks to Jahnel, an excellent stain with which we can really study spirochetes in general paralysis.

2. Spirochetes may be found in any part of the brain in general paralysis, but especially in the frontal parts. They are chiefly in the middle and deeper layers of the gray matter, rarely in the pia mater, almost never in the white matter, and are most often found in cases with a stormy clinical course.

3. Spirochetes may live for many hours after the death of the patient. They strongly resist decomposition, and may change their position postmortem. Therefore, necropsy should be performed as early as possible.

4. Spirochetes in general paralysis are either diffusely scattered or more rarely grouped in foci or swarms. They have no special relation to nerve cells or to other elements in nervous tissue, with the possible exception of blood vessels.

5. The time at which spirochetes enter the nervous parenchyma in general paralysis, and the pathways by which they spread after entrance, are not yet determined.

6. The spirochete of general paralysis is probably a syphilitic organism, altered biologically by various influences to which it is subjected during its long residence within its host. There is insufficient evidence that it is a special neurotropic strain different from the beginning. Experimental work with animals needs to be enlarged and well controlled.

7. Spirochetes are not absolutely limited to the nervous parenchyma in general paralysis; although not found in the general organs of the body, they are occasionally found in the mesoblastic tissues, pia mater and in the aorta.

8. The terms "parenchymatous neurosyphilis" and "interstitial neurosyphilis" seem hardly justifiable in the light of present studies.

9. Spirochetes have been demonstrated in the spinal pia-arachnoid in tabes.

10. A great field is open for further study along these lines, especially in experiments on animals.

11. Treatment of general paralysis need not be absolutely hopeless. Some of the spirochetes, at least, are probably accessible. The modified strain of spirochete in general paralysis with its hypothetical "heightened resistance" may need to be attacked by a modified therapeutic agent different from the one that succeeds with the ordinary syphilitic strains.

AN ANATOMIC STUDY OF THE FAISCEAU DE TÜRCK IN RELATION TO THE TEMPORAL LOBE*

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An opportunity for further study of the anatomic relations of the faisceau de Türck was afforded in a patient who was admitted to the neuropsychiatric wards of the Philadelphia General Hospital in my service on Nov. 30, 1920, and who died on Dec. 10, 1920. The clinical symptoms began six months before the fatal termination, but having no bearing on the anatomic study, will be omitted.

At necropsy examination, a tumor (Fig. 1) measuring 5 cm. anterio-posteriorly and 4 cm. transversely was found occupying the posterior two thirds of the first and second temporal convolutions. It was adherent to the dura, and a cross section showed marked vascularity. Posteriorly the tumor (Fig. 2) did not extend quite to the anterior occipital fissure. Macroscopically, in its posterior aspects, the tumor was rather sharply defined, but there was evidently some invasion of the adjoining cortex and white matter, that is, of the gyrus between the tumor and the anterior occipital fissure, to the extent of about 2.5 cm. Anteriorly, the tumor was less clearly defined except in its outer portion, where for a short distance beneath the cortex it was rather sharply outlined.

Dipping inward, anterior to the tumor, was a fissure which was believed to be the deep temporal fissure, anterior to which was what was regarded as the deep temporal convolution. This convolution macroscopically showed distinct implication by the tumor process. The tumor extended laterally into the brain tissue as far as the inferior longitudinal fasciculus and the optic radiations but did not appear to involve these bundles. Microscopically the tumor proved to be a glioma (Fig. 3).

The brain was cut in serial sections on both sides from the superior level of the tumor as far down as the pons. The tumor itself, with the

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* From the Neuropsychiatric Department of the Philadelphia General Hospital and the Neuropathological Laboratory of the Philadelphia General Hospital.

* Read at the Forty-Eighth Annual Meeting of the American Neurological Association, Washington, D. C., May, 1922.

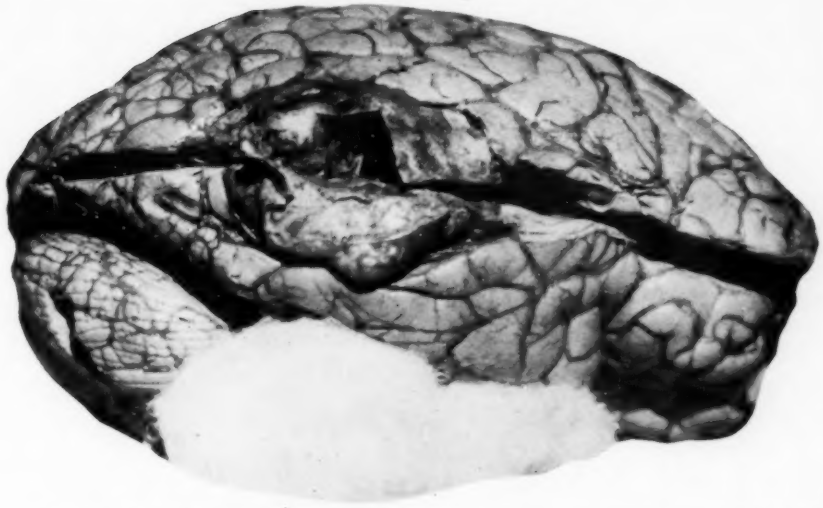

Fig. 1.—Tumor showing its relation to the posterior two thirds of the first and second temporal gyri.

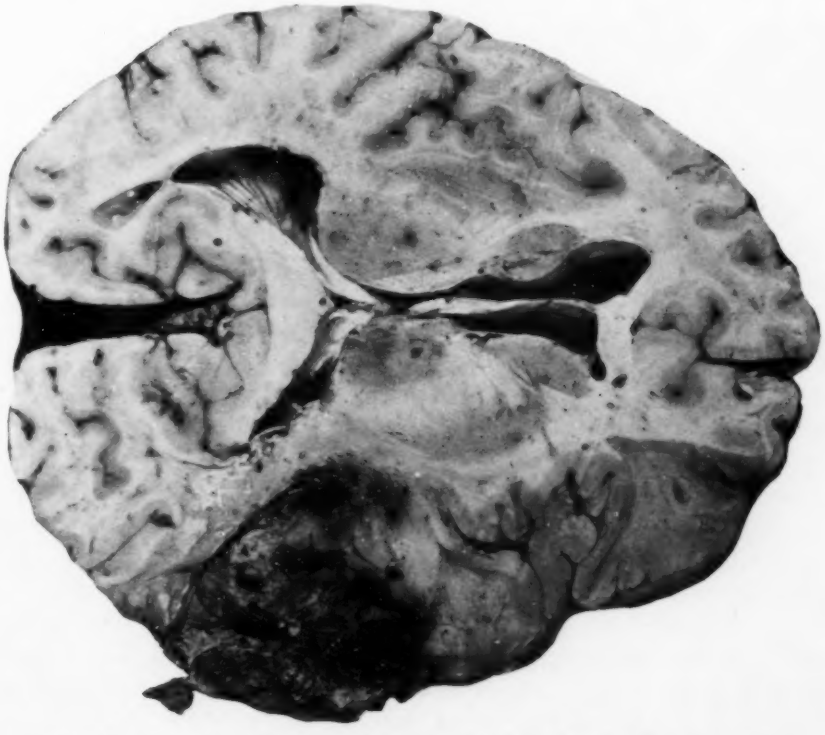

Fig. 2.—Cross-section of the brain showing the extent of the tumor.

adjacent brain tissue, was also cut at various levels in order to study its relation to the adjoining cortex and other brain structures. These sections were stained by the Weigert methods and by hematoxylin and eosin.

The cortex at the site of the tumor was completely destroyed, and nowhere in this region could any cortical tissue or white matter be discovered. The cortical and subcortical layers anterior to the tumor, namely, the deep temporal gyrus which dips normally down almost to the external capsule, the inferior longitudinal fasciculus and the optic radiations were differentiated as to gray and white matter, but in the tissue just adjoining the tumor there was pronounced cellular infiltra-

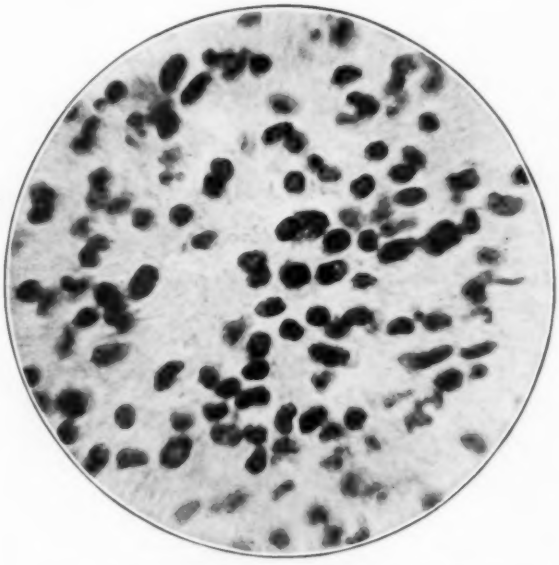

Fig. 3.—Microscopic section of the tumor.

tion with cells having the same characteristics as those found in the tumor.

Posterior to the tumor and between this and the anterior occipital fissure of Wernicke a similar condition could be observed, namely, differentiation of the cortical and white matter and implication of these by the cellular infiltration.

It was possible to conclude from this study that the cortex and white matter of the superior and middle temporal gyri were totally destroyed by the tumor, and the brain tissue anterior and posterior to these regions to the extent of about 2.5 cm. was implicated by a cellular infiltration consisting of cells similar to those found in the tumor proper.

A study of the serial sections of the brain internal to the tumor and the corresponding levels on the opposite side failed to reveal any evidence of degeneration on either side. The faisceau de Türck, at the point where it appears in the retrolenticular region, was intact. The foot of the peduncle stained uniformly and showed an undegenerated faisceau de Türck in this region (Fig. 4).

The duration of the tumor, which showed its first clinical manifestations six months prior to death, justifies the assumption that the degeneration should have had time to appear in the faisceau de Türck, where it appears in the retrolenticular region if not in the foot of the peduncle, if its cortical origin had been destroyed.

In 1912, I [1] read before the American Neurological Association a paper on a study of the faisceau de Türck; and I stated that the

Fig. 4.—Absence of degeneration of the faisceau de Türck.

anatomic relations of the faisceau de Türck were still a matter of dispute. The origin of this tract, according to Déjerine, was the middle portion of the temporal lobe, more especially the cortex of the second and third temporal lobes, which Kann and Brodman believed could be confirmed from their studies. The studies of Kattwinkle and Neumayer placed its origin in the third, second and first temporal convolutions. Flechsig and Van Gehuchten did not agree with this view. Von Bechterew, Flechsig and Meynert and others placed its origin in both the temporal and occipital lobes; Brero, in the parietal lobe; while von Monakow and others asserted that its origin was in the parietal and temporal lobes, von Monakow believing that some of the fibers came from the occipital lobe. Marie and Guillain, from a study of nineteen

1. Rhein, John H. W.: J. Nerv. & Ment. Dis. **38**: No. 9 (Sept.) 1911.

cases, believed that these fibers came from the third temporal convolution. The case of Mills and Spiller, in which the anterior part of the second temporal gyrus and a portion of its upper middle segment were degenerated without showing any involvement of the faisceau de Türck; the case of Löwenstein, in which the anterior half of the second temporal and the anterior two thirds of the temporal were implicated with an intact faisceau de Türck; and a case previously reported by myself in which there was atrophy of the middle portion of the second and third temporal and part of the first temporal convolutions without degeneration of this tract, led me to the conclusion at that time that it could originate only in the posterior part of the temporal gyrus, if at all in the temporal lobes. The case reported in this paper would indicate that the faisceau de Türck does not spring from the posterior two thirds of the superior and middle temporal gyri or the adjacent cortex.

A study of the literature cited demonstrates that the most reliable authorities exclude every portion of the cortex of the temporal convolutions except the posterior part of the inferior temporal gyrus as the origin of this fasciculus. From this, it may be concluded that the faisceau de Türck does not arise from any other portion of the temporal lobe since every other locality may be excluded as a possible origin. I know of no case in literature in which a lesion purely of the posterior portion of the inferior temporal gyrus has occurred and in which studies of the pathology have been made with a view to connecting the faisceau de Türck with this region.

The studies of nineteen cases by Marie and Guillain showed that the region most frequently giving rise to this degeneration is that portion just behind the posterior segment of the internal capsule in the white substance situated between the temporal convolutions and the external wall of the occipital horn of the lateral ventricle, where, they state, the fibers from the third temporal convolution pass. In the second case which I reported in 1912, the findings corresponded to those of Marie and Guillain. In this case, the lesion implicated the white matter of the posterior portion of the posterior segment of the external capsule as well as that of the posterior portion of the temporal lobe and the wall of the descending horn of the lateral ventricle, and the faisceau de Türck was almost completely degenerated. There was a cutting off of the fibers from the third temporal convolution as well as of those from the occipital lobe in part.

I think it may be concluded that if the temporal lobe is the origin of fibers which degenerate in a descending direction, consisting of the faisceau de Türck, they may come from the posterior third of the third temporal convolution.

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DISCUSSION

DR. ADOLF MEYER, Baltimore: It is very important for us to realize that the cases by which these matters can be brought to a test are relatively few. In the first place, the specimens of tumor are always somewhat hazardous on account of the fact that degenerations can be hidden or diffused. For a complete decision of the question we really need experimental material and operative extirpations such as occur occasionally. In that connection the temporal lobe, especially of the right side, may be sacrificed without serious damage. We also ought to use the cases of arteriosclerosis or embolic softening, which are not rare after all, but are unfortunately rarely referred to competent laboratories because most people think that it is old and long settled material. I have personally not had any material so far that appeared to me would throw conclusive evidence on this question. I have not obtained any embolism or softening within the Marchi period, and practically most of the specimens I have are fusions of temporal lobe and occipital lobe lesions.

Concerning the occipital lobe lesions I am absolutely sure of an efferent path to the pons. In a case published in the *Transactions of the American Physicians,* in 1905, an attempt at suicide led to an experimental destruction of the parietal lobe with destruction of the most dorsal parts of the sagittal marrows, with a very definite Marchi degeneration extending into Türck's bundle; but from the point of view of the temporal lobe I do not know of any American material at least that would be absolutely conclusive.

TUBEROUS SCLEROSIS*

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Tuberous sclerosis is a relatively rare developmental anomaly of the brain and other organs, characterized clinically by idiocy and epilepsy, and pathologically by multiple sclerotic nodes over the surface of the brain, multiple subependymal tumors, and multiple tumors of the heart, kidneys, skin and other organs.

Though the disease was described by several authors before 1900, von Recklinghausen reporting a case as far back as 1862, the minute histologic alterations and their significance were appreciated first in 1912 when Bundschuh [1] and Bielschowsky [2] independently gave us very complete and exact studies founded on the embryological concepts laid down by Ranke.[3] But little has appeared in English or American literature on the subject, although Campbell [4] described it in his work on cerebral sclerosis, and Sailer [5] collected twenty-eight cases in 1898. One American writer recently described a case as juvenile multiple sclerosis. It is important that these cases be recognized, for in addition to the epileptic seizures, these patients often present focal symptoms that may call for operation. It is the consensus of opinion, however, that operation has no favorable influence on their subsequent course.[6]

Because of the association of tumors of other organs with the focal sclerosis of the cortex, cases of tuberous sclerosis are recorded under many titles, and a comprehensive review of the literature is therefore difficult. Titular reference is in some instances to the cardiac tumors, as in von Recklinghausen's case; in others to the renal tumors; while

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* Presented before the Philadelphia Neurological Society, March 24, 1922.

* From the Laboratory of Neuropathology, Philadelphia General Hospital, and the Graduate School of Medicine, University of Pennsylvania.

1. Bundschuh: Ein weiterer Fall von tuberöser Sklerose, Ziegler's Beitr. f. path. Anat. u. allgem. Path. **54**:278, 1912.

2. Bielschowsky and Gallus: Klinische und anatomische Studien über tuberösen Hirnsklerose, J. f. Psychol. u. Neurol. **20**: Suppl. Erganzungsheft 1, 1913.

3. Ranke: Beitrag zur Kenntnis der normalen und pathologischen Hirnrindenbildung, Ziegler's Beitr. f. path. Anat. u. allgem. Path. **47**:51-126, 1909-1910.

4. Campbell, A. W.: Cerebral Sclerosis, Brain **28**:367-437, 1905.

5. Sailer, J.: Hypertrophic Nodular Gliosis, J. Nerv. & Ment. Dis. **25**: 402, 1898.

6. Volland: Untersuchungsresultate von 50 Schädeltrepanationen bei Epilepsie, Ztschr. f. d. ges, Neurol. u. Psychiat. **74**:505, 1922.

still others, in which the cutaneous tumors were prominent, are found under the name of adenoma sebaceum. On an estimate, about 100 cases have been reported.

ETIOLOGY

Degeneracy in the parents is an etiologic factor. The disease is congenital, but neither familial nor hereditary. Apparently it begins between the fourth and seventh fetal month. Ranke and Bundschuh have pointed out that the primary and secondary fissures which are completed about the fourth month are not distorted by the sclerotic patches, while the tertiary convolutions which develop later are involved. Bundschuh further called attention to the persistence of an external granule layer over the sclerotic areas which disappears in normal development about the seventh fetal month, and which is never present at birth.

CLINICAL COURSE

The disease is characterized clinically by idiocy and epilepsy. Convulsions usually are the first manifest symptom. In Hartdegen's case the attacks began a few hours after birth and continued until the end, two days later. Brückner's patient, on the other hand, was 9 years old when the first convulsion occurred. When once initiated the convulsions usually continue, although they may decrease in frequency or even cease. The first attacks are usually mild, without loss of consciousness, and they may affect only isolated groups of muscles. During a period of years, however, they become gradually more severe and may even be the direct cause of death. The attacks are quite evenly spaced, are not so frequent as genuine epileptic attacks, nor so paroxysmal as those of the atrophic scleroses. At times equivalents of attacks are noted, fainting spells, maniacal attacks and so-called psychic epilepsy.

In cases in which the chief clinical expression is idiocy, development may be comparatively normal for several years. Usually the children are slow to walk or talk, but sometimes they are active and intelligent and may even attend school for a number of years. More characteristic than the cessation of development, however, is the retrograde process which follows it. The child ceases to take interest in playthings, prefers to sit still, loses the power of attention, becomes indifferent to everything which had previously attracted it, becomes untidy and often masturbates excessively.

Focal signs, such as localized pareses and contractures, muscular spasms and speech defects, are frequently present. Bielschowsky has reported a case in which movements on one side of the body were much retarded, with rigidity and tremor limited to that side. He records an area of sclerosis in the opposite basal ganglion.

Although status epilepticus is sometimes the direct cause of death, the patients usually die of intercurrent infection, especially of tuberculosis, or of gastro-intestinal disturbances. Occasionally the renal tumors, becoming excessively large, cause death. Patients with cardiac tumors usually die young.

DIAGNOSIS

Often the diagnosis cannot be made during life. When epilepsy and idiocy appear in a child, especially when they are progressive and when localizing signs appear, tuberous sclerosis should be considered. These signs are uncertain, however, and only when tumors of the skin are present is the diagnosis justifiable. In the Pringle[7] type these tumors grow on the face, are small, firm papillary growths, pale to dark red, vary in size up to 1 cm., and have a "butterfly" distribution over the nose and cheeks. The Barlow type is characterized by larger nodes, sometimes 2 cm. in diameter, usually found on the trunk. They are adenomas of the sebaceous glands. Sometimes there are abnormalities in the growth of the hair. The cutaneous lesions often appear first at adolescence, giving no assistance in diagnosis during the early years of life.

Rarer malformations occasionally found are tumors of the duodenum, spleen and liver; imperfections of the heart, such as patent ductus arteriosus, cardia trilocularis, origin of the aorta from both ventricles; ectopia testis, etc.

REPORT OF A CASE

History.—M. D., Philadelphia General Hospital, service of Dr. Weisenburg, aged 6 years. The father was 29 years of age, the mother 24 at the time of conception. Both were mentally healthy and of good inheritance, but the father was tuberculous and died two months before the child was born. Birth was natural and at full term; the patient was a well developed female child. She was breast fed until 9 months of age. At seven or eight weeks of age she began to have tonic convulsions lasting about two minutes followed by stupor of several minutes' duration. The convulsions increased in frequency for six months, then decreased and ceased at the age of 5 years. During the eleven months she was in the hospital no convulsions were recorded.

Although well formed, the child was backward, could not sit up until she was 2 years old, never learned to walk, talk or feed herself; she did not play with toys until she was 4. Her chief occupation was tearing up her clothing and putting the pieces in her mouth. She masturbated constantly. She could see and hear, but apparently recognized nobody, not even her mother. She would stop tearing her clothes when spoken to, but almost immediately resume it. She was large for her age. The head was slightly enlarged; the skin was without blemish; muscular power and coordination were good. Patellar reflexes were exaggerated. Urine, blood count and spinal fluid were normal. The Wassermann test on the blood and spinal fluid was negative.

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7. Pringle: A Case of Congenital Adenoma Sebaceum, Brit. J. Dermat. **1**:64, 1891.

Before being brought to the hospital, and during her stay there she had occasional attacks of vomiting immediately after taking food, although her appetite was excellent. She became emaciated, and finally died of inanition Jan 21, 1921. She was then 6 years old.

Clinical Diagnosis: Imbecility, hydrocephalus, enteritis.

Necropsy Record (Dr. Morton McCutcheon).—The body was that of a white girl 6 years of age, weighing 40 pounds (18.14 kg.). Bony development was normal; there was marked emaciation; rigor and livor were present. The skull was slightly enlarged and normal in shape; the pupils were equal and regular, the sclerae clear. The thorax was symmetrical, the abdomen retracted. Extremities, external genitalia and hair distribution were normal.

The thoracic organs were normal. The spleen weighed 40 gm., and was normal, as were the stomach and intestines. The liver weighed 540 gm. and was of normal size and consistence. The lower border was rounded. The cut surface showed a number of yellowish white lobules, the other lobules being brown. The discolored areas did not bulge. The pancreas was normal.

The left kidney weighed 60 gm., and was of normal size and consistence. The capsule stripped readily leaving a smooth pinkish gray surface. Striations were normal. Several pale yellow nodules were present in the cortical substance, the largest being 4 mm. in diameter. Some of them bulged distinctly on section although they could not be lifted out, and one bulged through the capsule. The consistence of these structures was that of normal renal substance. The right kidney weighed 50 gm. and resembled its fellow. The suprarenals, aorta, ureters, bladder and internal genitalia were normal.

The brain was distinctly enlarged; it weighed 1,120 gm. The calvarium and dura were normal. The pia was slightly opaque in places but not markedly thickened; it was nowhere adherent to the cortex. The frontal poles of the brain were smaller than usual and unnaturally white. They felt as hard as the normal brain after fixation in liquor formaldehydi. In the temporal, parietal and occipital lobes there were similar hard areas in which the convolutions were larger than normal, projected above the general contour, were flattened or even umbilicate on the surface and unnaturally pale. These areas were fairly sharply circumscribed, irregular in outline and bounded almost everywhere by tertiary fissures. The overlying pia was lightly attached. Except for a small area in the right paracentral lobule, the central convolutions had escaped. Hippocampus, cerebellum, brain stem and cervical cord were normal.

After fixation in 10 per cent. liquor formaldehydi, the usual section was made through the basal ganglions (Fig. 1). The hypertrophic convolutions were broader at the surface than at the base and compressed the normal gyri which lay between them (Fig. 2). The cortex of the sclerotic area varied considerably in thickness; it was distinguished from the underlying white matter, not so much by color as by difference in texture, the cortex being densely hard and the white matter porous. In the fissures the tissue was softer than on the convexity. In the white matter beneath some of the sclerotic gyri were minute areas resembling the overlying cortex. Microscopic examination proved these to be heterotopias of gray matter.

In the right frontal pole, where the area of sclerosis was most extensive, there was a multilocular cyst measuring about 1 cm. in diameter, without softening, pigmentation or obvious degeneration in the neighborhood.

The posterior horn of the left lateral ventricle measured 32 mm. across. Its external surface underlay a large sclerotic area in the temporal lobe. The cerebral tissue was here reduced to 11 mm. At the point chosen for study the

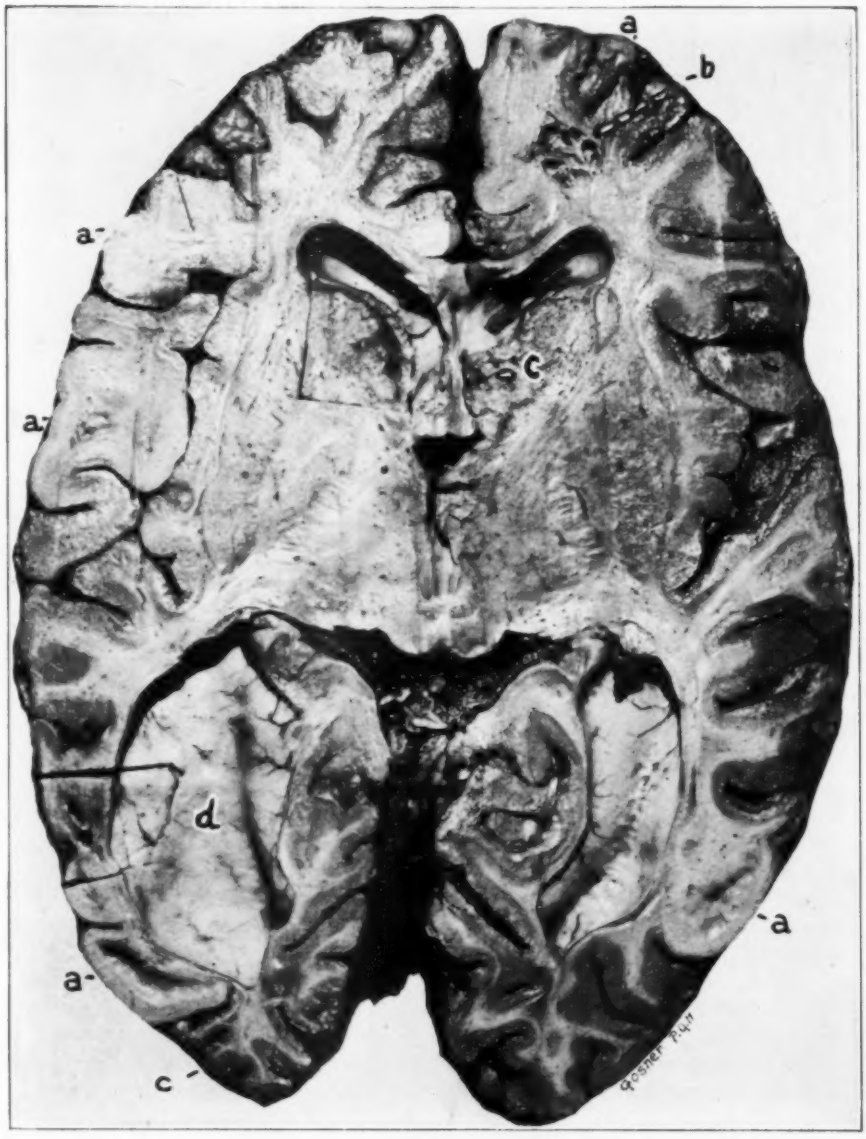

Fig. 1.—Horizontal section of brain: *a*, sclerotic areas of pale color and dense consistency with depression of underlying white matter; *b*, multilocular cyst in right frontal pole formed by dilatation of perivascular lymphatic spaces; *c*, tumor at head of caudate nucleus pressing into foramen of Munro; *d*, dilated posterior horn of lateral ventricle; note the area of microgyria in left occipital lobe.

cortex was narrow, white and hard, and the white matter was soft and porous except where the fibers of the optic radiation ran, skirting the horn of the ventricle. This tract was narrow, but apparently in good condition.

In the right parietal lobe over an area 3 by 4 cm. there were numerous abnormally small and branched convolutions, in which scarcely any white matter could be seen underlying the narrow cortex. The area was not depressed below the general level as is usual in microgyria. While these convolutions were of normal consistence and relatively normal architecture, there was a sharply defined area of sclerosis immediately adjacent. A similar area appeared in the left occipital pole.

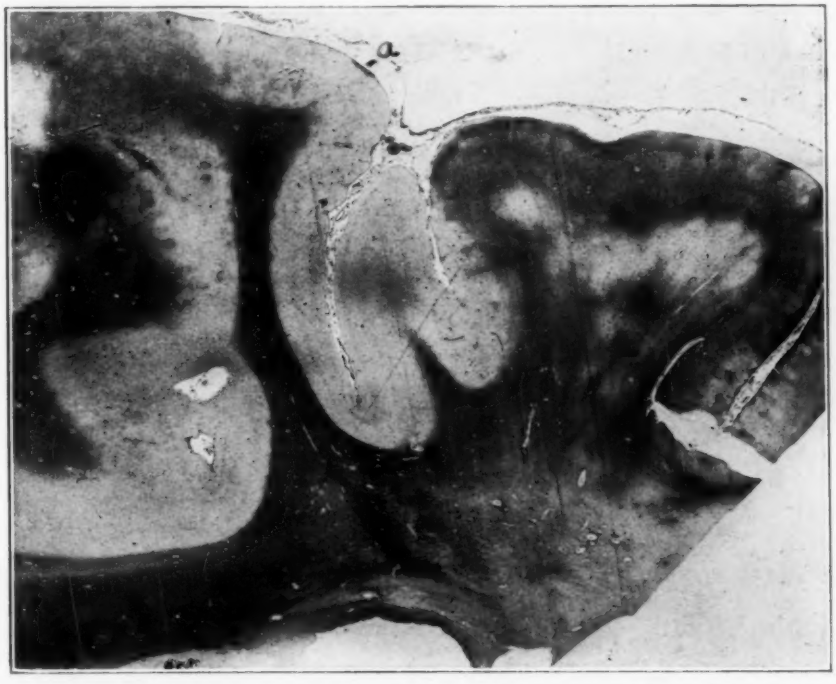

Fig. 2.—Hypertrophic convolution, broadened and umbilicate at surface, due to neuroglial overgrowth. The gliosis diminishes at the bottom of the fissure. Phosphotungstic acid-hemåtin stain; *a*, patch of beginning sclerosis.

At the head of the caudate nucleus on the right there was a tumor measuring 18 by 10 mm. pressing down into the foramen of Monro (Fig. 1). The tumor sprang superficially from the caudate nucleus, was of soft consistence, rather friable and darker than the surrounding tissue. It was circumscribed though not encapsulated. Its surface in the ventricle was fungoid and irregular, but at the sides it was smoother and apparently covered by ependyma. It did not invade the nervous tissue. At other points on the surface of the caudate nucleus on both sides there were smaller tumors varying from 2 to 6 mm. in diameter. In the third ventricle there were tumors up to 5 mm. in diameter, especially along the striae terminales. These smaller tumors occurred singly and in groups, and between the larger ones the ependyma was raised into cords.

Where a single tumor rose the ependyma was arranged in radial cords, resembling buttresses. The smaller tumors were much harder than the surrounding tissue and grated on the knife on sectioning. Between the tumor and the normal basal ganglion there was a layer of pale, firm tissue encapsulating the growth. In the vicinity of the tumors granular ependymitis was visible (Fig. 3). The fourth ventricle contained no tumors.

Gross Findings.—The gross findings corresponded closely to the descriptions given by Pelizzi, Vogt and Bielschowsky. They describe two varieties of tubera: the hypertrophic convolution and the sharply circumscribed node which has no definite cortical characteristics. These exist in varying proportions in different cases, but usually both are present. I could find none of the circumscribed nodes. Broadening of the convexity of the convolution and dimpling in the center were described by older authors, but Pelizzi was the first to show that the sclerotic process was confined to the external surface of the convolution and seldom or never reached the bottom of the fissure, even where two densely sclerotic gyri lay adjacent.

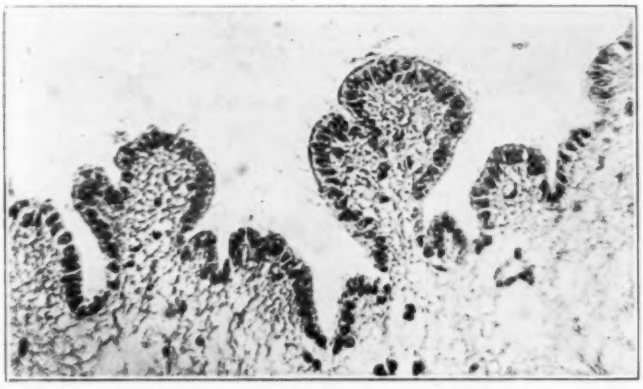

Fig. 3.—Granular ependymitis in vicinity of subependymal tumors.

The cyst of the frontal pole was unusual. Bundschuh found symmetrical cysts in the occipital poles in his case, and others have described them. Localized microgyria was described by Bielschowsky. Ventricular tumors were encountered as frequently as the cortical scleroses and did not vary much from type. They were usually hard, nearly spherical, lay immediately beneath the ependyma and varied in size up to 8 mm. Their usual locations were the caudate nucleus, striae terminales and thalamus, occasionally the fourth ventricle. On account of the persistent and uncontrollable vomiting without evident local cause, the fourth ventricle was examined with particular care but no abnormalities were found. The tumor at the head of the caudate nucleus was exceptional in size and general characteristics. Whether the hydrocephalus was the result of obstruction of the foramen of Munro by this tumor is not certain. Hydrocephalus is present in a minority of cases, and without known cause. The third and fourth ventricles were not dilated.

MICROSCOPIC EXAMINATION

Ranke [3] as a working hypothesis to explain the pathology of tuberous sclerosis suggests that there are two stages of differentiation of cells

of the central nervous system from germinal cells to fully developed ganglion cells and neuroglia cells. First, the cells develop to the point of differentiation when they become either neuroblasts or spongioblasts, and then they ripen into nerve cells and glia cells. This he acknowledges is purely theoretical, since none of the present histologic methods differentiate future neuroblasts from future spongioblasts. If the cells are disturbed at the time of differentiation, a great variety of forms may be produced. "Three extremes are thinkable: first, that the disturbance would result in the production from indifferent elements of nerve cells alone; second, of glia cells alone; and third, that no differentiation would take place. The first two possibilities are extreme one-sided differentiation, and the last, extreme lack of differentiation." Ranke elaborates the theory further by suggesting that harmful influences acting on the neuroblasts and spongioblasts at various periods of their development might produce further anomalies, and also that various combinations might occur.

The so-called "large cells" so characteristic of tuberous sclerosis are believed to be incompletely differentiated neuroblasts. They show the following anomalies (Fig. 4):

(*a*) Position: Some lie next to the surface; many are distributed through the cortex; some lie in the white matter, either singly or in heterotopic groups. They are seldom arranged in laminae.

(*b*) Size: The cells equal or exceed large pyramidal cells in size.

(*c*) Form: Occasionally a cell is seen that would be termed a ganglion cell were it not for its size or location. This is rare. More often the cell is round or oval, occasionally spindle-shaped or snakelike.

(*d*) Processes: There may be no processes or many processes, and they vary markedly in size. The large round cells have no processes as a rule, but some of them have scores resembling a Medusa's head. The spindle cells have two, and sometimes they dwarf the cell by comparison, extending full across the high power field, dividing, and wandering through the layers of the cortex (Compare Figs. 4, 5, 6).

(*e*) Nucleus: Many of the round cells have no nuclei. When present the nucleus is eccentric. Often it is lobed and sometimes two, three or more nuclei may be seen in a single cell. Some nuclei are small, but usually they are large; often grotesquely distorted; always pale and vesicular. Nucleoli are usually absent, and the chromatin network is very loose.

(*f*) Internal Structure: The round cells have neither extranuclear granules nor neurofibrillae, but the snakelike cells have fibrillae in their processes although seldom in the cell body. Sometimes suggestions of tigroid bodies are found in these more perfectly formed elements.

Investigators do not agree that these "large cells" are of neuroblastic origin. Several authors, especially the earlier ones, class them as

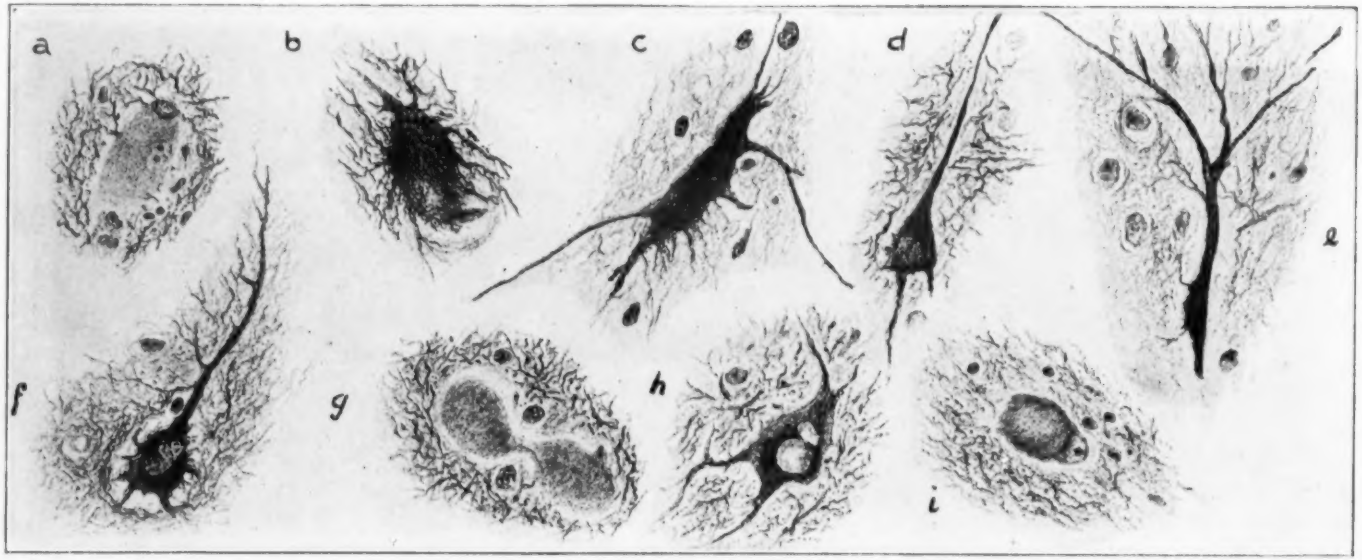

Fig. 4.—Various types of faultily differentiated cells of neuroblastic origin: *a*, simple undifferentiated "large cell"; *b*, Medusa cell; *c*, large grotesquely differentiated ganglion cell; *d*, normal ganglion cell of large pyramidal type; *e*, snakelike cell; *f*, atypical ganglion cell; *g*, twin cell; *h*, "large cell" with bilobate nucleus from heterotopia; *i*, large cell with polar nucleus. Silver diffusion stain; × 1,000.

neuroglia cells. Bielschowsky indicates their neuroblastic origin by pointing out gradations from normal nerve cells to round undifferentiated ones on the one hand and snakelike ones on the other. Their staining reactions and the presence of neurofibrillae as demonstrated by Alzheimer also indicate this origin.

Fig. 5. — Low-power view of sclerotic cortex showing neuroglia margin, external granule layer, and atypical "large cells." Silver diffusion stain.

While these cells occurred usually in sclerotic areas, they were found also in the presumably normal cortex nearby; and they were more numerous in the deeper cortical strata and in the white matter than on the surface. The heterotopias consisted of groups of "large cells" interspersed with a few glia cells and fibers, and more or less perfectly formed nerve cells (Fig. 6).

NEUROGLIA

The density of the sclerotic cortex was due to innumerable glia fibers that intertwined in every direction. At the surface the fibers were sometimes collected into sheaves and bundles and stood on end above the normal margin (Fig. 5) but did not invade the pia. They were everywhere abundant, forming

a band many times the thickness of the normal "Randglia" (Fig. 5). At the surface of otherwise normal cortex, the first sign of sclerosis was thickening of the external glia margin and increase of glia nuclei, with sometimes a few large undifferentiated cells (Fig. 7).

Of more interest from the point of view of development was the external granule layer present in the most densely sclerotic areas. Beneath the broad surface layer of neuroglia lay a stratum of round cells, evidently glial in nature, divided from the deeper lying strata of nerve cells by another band of neuroglia fibers (Fig. 5).

Throughout the sclerotic areas there was a dense feltwork of glia fibers in the meshes of which lay numerous small nuclei. Most of these were in the

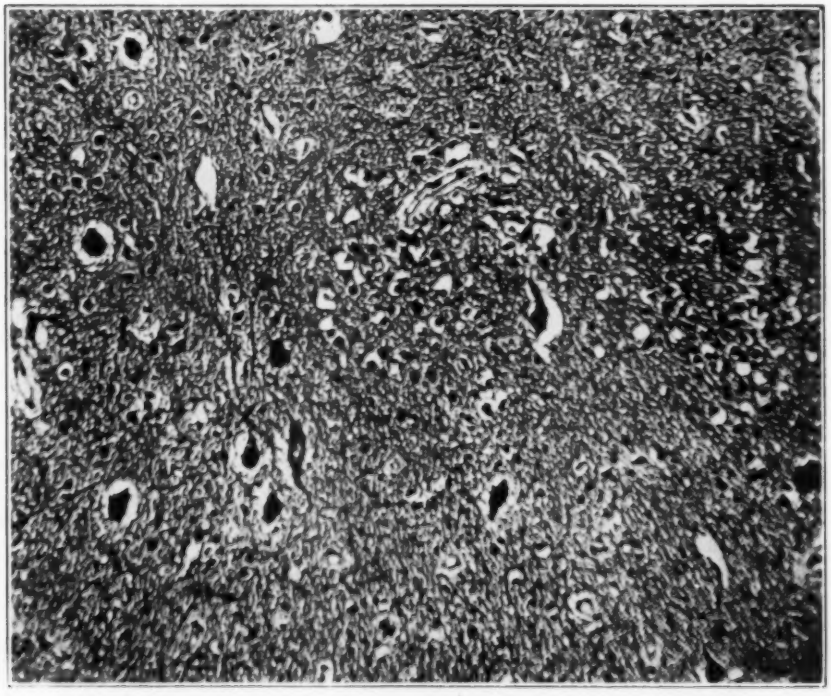

Fig. 6.—Higher power view of one of the heterotopias. Numerous atypical small nerve cells, some large grotesquely differentiated nerve cells and moderate gliosis.

resting stage but many were fiber formers. Where the feltwork was dense, it was impossible to trace the individual processes; but in the white matter where the tissue became spongy, the glia feltwork was much looser, and here the individual cells stood out with greater prominence (Fig. 8). They were larger than normal and possessed fairly definite cell bodies. The fibers were deeply stained, seemed to spring from the periphery of the cell, were heavy, and at a short distance from the cell divided dichotomously into larger and smaller branches which then curved away in easy paths and broke up into the usual glia fibers.

In the hypertrophic convolutions typical nerve cells were few, small and not arranged in strata; their apical dendrites pointed in various directions, and

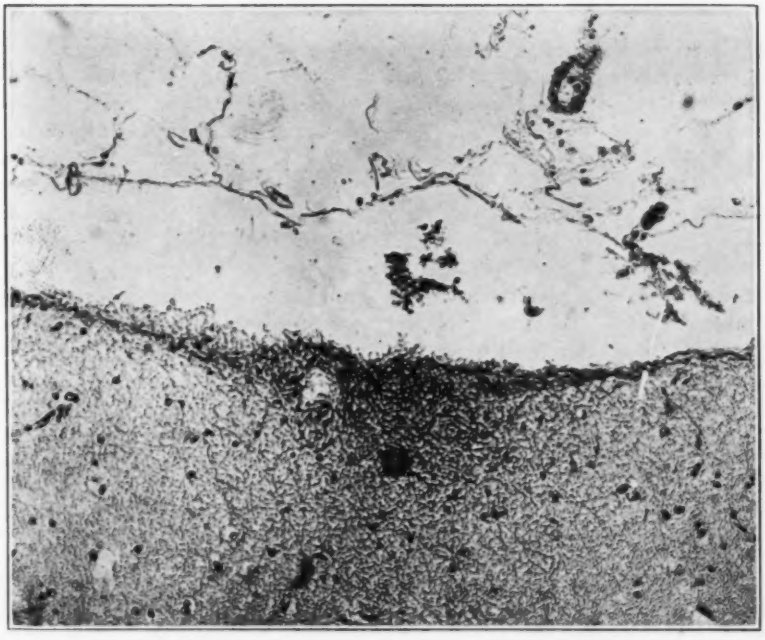

Fig. 7.—Earliest sign of beginning sclerosis. Increase of marginal glia associated with a few large undifferentiated cells. Neuroglia stain.

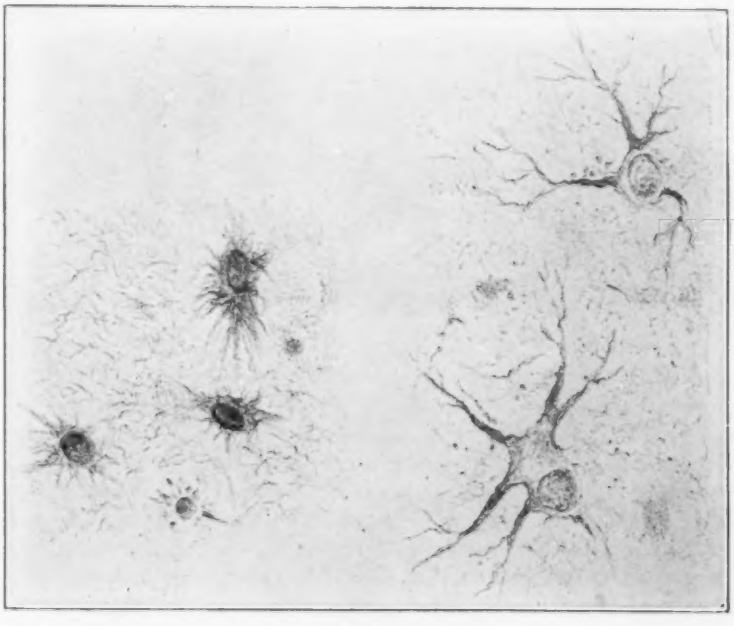

Fig. 8.—Neuroglia cells from loose-meshed white matter; very many processes extending from all points of cells; giant neuroglia cells from junction of white and gray matter; × 1,800.

they contained only remnants of internal structure. Myelin sheaths were almost entirely lacking. By silver impregnation, however, a surprisingly large number of nerve fibers was demonstrated even in the most densely sclerotic areas and the underlying loose-meshed white matter. Moreover, there seemed to be little interference with the projection tracts, for the spinal cord showed no degenerative changes, and such tracts as the optic radiation were well preserved.

Scarcity of myelin sheaths was quite as characteristic as overgrowth of neuroglia in the hypertrophic convolutions, yet in one area studied, nerve fibers appeared to be surrounded by multiple concentric rings, sometimes irregular or oval, depending on the angle of the section. These stain poorly for myelin, but clearly with phosphotungstic acid. They appear to be hyperplastic but poorly differentiated myelin sheaths.

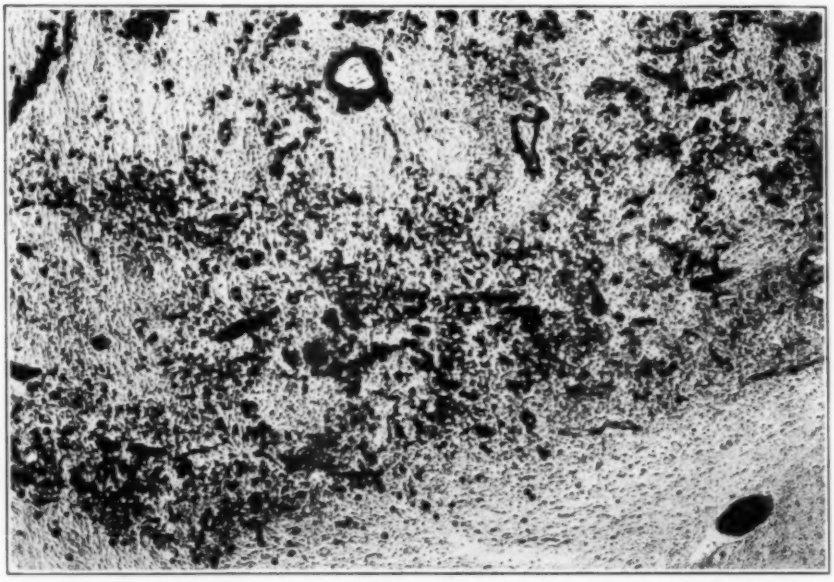

Fig. 9.—Concretions in old compact tumor. They appear to originate from blood vessels.

SUBEPENDYMAL TUMORS

All the tumors in this case except the large soft one were covered by ependyma. They arose from the subependymal glia layer and projected into the ventricle. The process of tumor formation here could be seen by examining the wall of the ventricle. The subependymal neuroglia layer became thicker, the cells developed processes, and a few large undifferentiated cells appeared, thus paralleling the picture seen in the cortical scleroses. At first the tumor was covered with ependymal cells, but as growth proceeded this layer became much flattened and rupture might have occurred so that the ependymal covering was lost. The tumors which had broken through the ependyma were more vascular and apparently of more rapid growth.

The early tumor is merely a collection of large cells with neuroglia fibers running between cells and surrounding it. In the larger tumors the cells are

more numerous and the glia fibers are proportionately diminished. They separate it into cell groups by trabeculae, and wall it off from the underlying ganglion. Many vessels in these moderate sized tumors are infiltrated with calcium salts (Fig. 9). There is granular ependymitis in the vicinity of the tumors (Fig. 3). Whether the cells of these subependymal tumors are of nervous or supporting origin is contested, some authors considering them as large cell gliomas, others as ganglioneuromas, and still others as neuroblastomas. The majority ally them with the gliomas.

The typical cells of these tumors are fusiform or retort-shaped, vary in size up to that of the largest of the Betz cells, have no tigroid bodies or neurofibrillae, and send processes away from one or both ends. Some of the

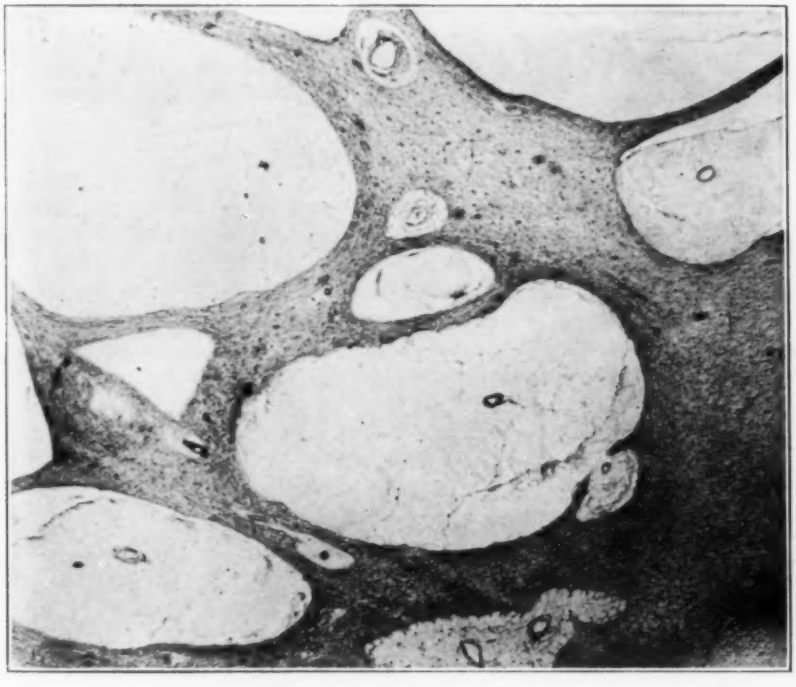

Fig. 10.—Low-power view of multilocular cyst.

cells are almost round and have no processes. The nuclei are usually large, with definite nuclear membrane, delicate chromatin network, and only occasional nucleoli. Sometimes the nuclei are grotesque in size or shape, or two or more nuclei are found in the same cell. Mitotic figures are absent. Many of the larger cells have vacuoles but otherwise do not seem to be degenerated.

The fibers sent off by the large cells run in bundles and separate the cells from one another into groups. Among these fibers lie cells of a different character, usually much elongated, with large distorted nuclei and processes following the cell outline. These take the glia stain typically whereas the large cells stain like ganglion cells.

The large tumor in this case varied in microscopic as well as gross characteristics from the others. The cells were not divided into groups, and glia

cells and fibers were almost absent. The cells were polygonal or spherical and had no processes. They stained more heavily than those of the smaller tumors, were smaller in size, and twin cells and multinucleated cells were more frequent. The extreme edge of the tumor was serrated with cell columns projecting into the ventricular lumen. There was a rich supply of vessels of large caliber and thin walls. In many respects the tumor resembled a malignant growth.

BLOOD VESSELS AND CYST

Many of the pial vessels were thickened by overgrowth of the middle coat, and the elastic lamina was split into two or more layers. There was no perivascular round cell infiltration or other sign of inflammation.

In the cortex of the sclerotic areas the blood vessels were less numerous than usual and many were abnormally thick-walled. The perivascular spaces were increased. In the white matter this increase became striking and was of two forms. In the first the adventitia was increased, filling up the space and giving the vessel the appearance of an enormously thickened wall, although the tissue was loose-meshed and nuclei were infrequent. In the other form there was further dilatation of the space to enormous proportions, without corresponding increase of adventitia, thus forming cavities (Fig. 10). This was probably the origin of the multilocular cyst of the frontal pole. Beneath a densely sclerotic cortex appeared large spaces, vacant except for an extremely delicate meshwork of fibrous tissue surrounding a small blood vessel that ran through the cavity. Between the various cavities ran trabeculae of nerve and glia fibers, with many glia nuclei in the resting stage. These septums varied in thickness down to some so fine that they were formed by only a dozen glia fibers. Moreover, in the walls of the larger spaces there were projections of glia fibers, remnants of septums which had ruptured and retracted. These cysts had no lining, and there were no degenerative changes in the adjacent tissues. They were apparently enormous dilatations of perivascular lymph spaces brought about by disappearance of nervous tissue.

CONCRETIONS

Concretions were numerous in the subependymal tumors. In the sections some of these elements were circular, intensely black spots; others had central areas of lighter color; others had lumina containing red blood cells, and still others were obviously calcified capillaries and small blood vessels. This finding has been recorded by many investigators who agree that some vessels show calcareous degeneration, but the great number and uniformity of these masses in some places has led to the suggestion that some of them are calcified nervous elements. Calcium infiltration was observed in certain large cells in the sclerotic areas. These granules were most numerous in old compact tumors with dense glia capsules and few functioning vessels. On the other hand, there was no generalized degeneration of cells, and some apparently perfect tumor cells were seen lying next the calcareous bodies. Ranke ascribes both a vascular and cellular origin to these bodies.

RENAL TUMORS

These varied in character, some consisting almost entirely of fat, others of spindle cells, others of thick-walled blood vessels. Occasionally all these

elements were found in a single tumor. They were inlaid in the renal parenchyma, compressed it but little, were sharply circumscribed but not encapsulated, and gave no hint of vegetative activity.

There were no sebaceous or cardiac tumors.

SUMMARY

The cerebral changes in tuberous sclerosis are:

1. Abnormal differentiation of germinal cells during the middle fetal months with the production of neuroglial cerebral sclerosis and subependymal tumors.

2. Incomplete differentiation of ganglion cells with the production of bizarre types.

3. Persistence of the external granule layer of the cerebral cortex.

4. Agenesis of myelin sheaths in the sclerotic areas and underlying white matter.

5. Calcareous degeneration of the walls of vessels in the subependymal tumors.

6. Localized microgyria.

In addition, in this particular case there was dilatation of the perivascular lymph spaces with cavity formation. There was no involvement of the projection tracts.

DISTURBANCES OF THE RESPIRATORY RHYTHM IN CHILDREN

A SEQUELA TO EPIDEMIC ENCEPHALITIS

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The manifestations of epidemic encephalitis in children have received attention in this and other countries, and it has been our privilege in the Mayo Clinic to see nearly all the features described by other observers. However, one syndrome in children, which was presented to us more forcibly than the other more common disturbances of function, was a disturbance of the respiratory rhythm and depth, persisting many months after the initial phase of the disease had passed. Seven children and one adolescent came to the Mayo Clinic during the last half of 1921 and the first half of 1922, manifesting, besides the characteristic disturbance of the respiratory function, the now well recognized intractable insomnia and metamorphosis of character seen in children who have had epidemic encephalitis. Six of the patients dated their illnesses from the first three months of 1920.

Disturbances in respiration during the acute stages of encephalitis have already been described by Happ and Mason;[1] six of their eighty-one patients had marked hyperpnea during the early part of their illness. In the 115 cases analyzed by Dunn[2] there were three patients who had hyperpnea lasting for a few days. Epidemic hiccup was a form of respiratory disturbance associated with encephalitis, and later events showed that hiccup might be the sole manifestation of an abortive type. Sicard and Paraf[3] reported the cases of a series of patients who had suffered from various paroxysmal respiratory disturbances in the form of hiccup, yawning, stretching, sobbing and uncontrollable laughing. Aronson[4] observed a boy, aged 8, who during his convalescence from encephalitis had developed a protracted hyperpnea which had lasted for seven weeks. Hass described the respiratory

1. Happ, W. M., and Mason, V. R.: Epidemic Encephalitis. A Clinical Study, Bull. Johns Hopkins Hosp. **32**:137, 1921.

2. Dunn, A. D., and Heagey, F. W.: Epidemic Encephalitis: Including a Review of 115 American Cases, Am. J. Med. Sc. **160**:568-582, 1920.

3. Sicard and Paraf: Fourire syncopale et baillements au cours de l'encephalite epidemique, Bull. et mém. Soc. méd. d. hôp. de Paris, **45**:232-234, 1921.

4. Aronson, L. S.: Encephalitis with Unusual Sequelae, Neurol. Bull. **3**: 113, 1921.

disturbance of a child, aged 2 years, who, without any initial febrile episode, developed insomnia and change of character. The child suffered from attacks of forcible breathing followed by breath holding, cyanosis and unconsciousness. She had had as many as six attacks in one hour. In recent literature cases of disturbed respiration and bizarre performances have been reported.

In nearly all our patients the initial febrile attack was mild; the more severe symptoms followed later in some cases. This is in accord with the experiences of others, Marie and Levy [5] in particular, who gave the name of tardy encephalitis to this type of the disease. The persistency of the syndrome following so slight an onset left, however, no doubt with regard to the nature of the disease. It seems characteristic of the disease in children that the onset is mild, but the sequelae are none the less severe.

REPORT OF CASES

Case 1.—C. P., a thin undernourished boy, aged 14, was brought to the Clinic, May 9, 1921. In January, 1920, he had suffered from pains along the left sciatic nerve, followed by myoclonic jerking of the left lower extremity. Marked insomnia, polyuria and polydipsia had developed, and he had become dull and had lost interest in his studies. Later the right side of his body had become slow, stiff and clumsy. In May, 1921, grunting expirations had developed, and later stretching and bending motions of the trunk and limbs. These had persisted and insomnia had increased.

During a paroxysm the child suddenly rose from his seat and stretched his body so that his back was strongly curved forward and his abdomen protuberant. His chin usually was sunk on his chest and his arms rotated outward in the attitude of an early morning stretch and yawn. He held his breath in this position for about twenty seconds, then dropped back on the seat, thrust his head between his knees, and in a doubled position released his breath in a series of coughing grunts. These attacks might be repeated again and again day and night; in fact, the boy scarcely slept (Fig. 1). The boy had definite nystagmus, horizontal and vertical, loss of speed, and clumsiness in his whole right side. He had a bilateral Babinski sign and definite parkinsonian facies, gait, and posture. He drooled saliva, and he had slight dysarthria and tremor of both hands.

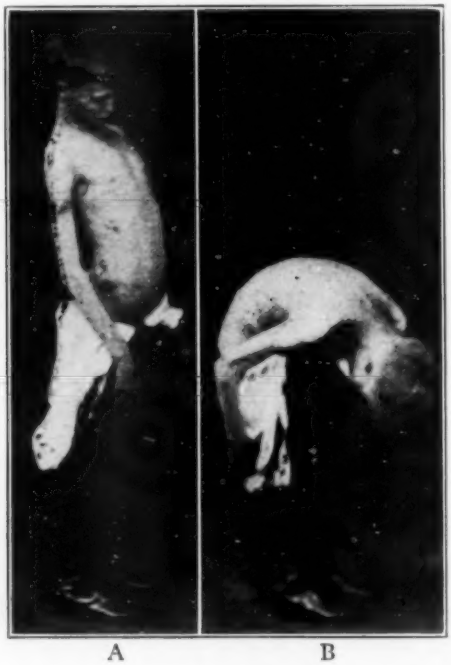
While the child was under observation for two months, the movements were continued and all sorts of therapy including hypnosis yielded no positive results. He was dismissed somewhat improved, but two months later his parents reported that he was as bad as before.

Case 2.—O. K., a girl, aged 12, was brought to the Clinic, Jan. 18, 1922. In March, 1920, she had had what was diagnosed as influenza and had been in bed for four days with a high temperature, headache and general malaise. Marked insomnia with choreiform movements followed; her disposition changed, and she became impulsive, stubborn and disobedient. Often during 1920 she had stayed up all night at her studies, but had made less progress than before. In the first few months of 1921, she began to breathe noisily and rapidly, and

5. Marie, P., and Levy, G.: Le syndrome excito-moteur de l'encephalite epidemique, Rev. neurol. **36**:513-539, 1920.

she later supplemented this with breath-holding attacks in which she stood erect with her head thrown back. By the end of 1921, these attacks became so severe that she lost consciousness at the end and fell, hurting herself on more than one occasion. The attacks often occurred at night, and the patient had to be watched to prevent serious injury.

During an attack the girl suddenly stood erect in the "attention" position with chest fully inflated, head thrown back, arms stiffly at her sides, and hands clinched. She held her breath in this position for from twenty to thirty seconds and became cyanosed, so that when she finally released her breath she staggered or fell, breathing noisily in deep quick pants (Fig. 3). After a few seconds, the head jerked quickly backward again. This was continued until the child became dull and stupid, and sat down unsteadily, breathing imper-



A B

Fig. 1 (Case 1).—*A*, attitude of patient at the height of the respiratory phase; complete cessation of breathing for twenty seconds; *B*, attitude at the end of the expiratory phase. The patient is releasing his breath in a series of coughing grunts.

ceptibly. Later the attacks were repeated for an indefinite period. In intervals between attacks the child breathed noisily, and was disobedient, impulsive and explosive in her speech. She is still under observation and has shown no improvement.

Case 3.—M. C., a girl, aged 2 years and 10 months, was brought to the Clinic, Sept. 14, 1921. After an attack of tonsillitis in January, 1920, she had slept badly, had become backward in development, had ceased to talk or play with other children, and was irritable, fretful and peevish. June, 1920, she awoke one night and went into a series of breath-holding attacks, but the

next day she seemed as usual; every night thereafter these attacks were resumed. Two months before, the attacks had occurred during the day as well. All correction or admonition was useless; even beating during an attack was of no avail, as she did not seem to feel the blows. A few days before, a continued series of attacks had occurred from midnight until 11 p. m.

Fig. 2.—Attitude of patient during the height of the inspiratory phase. Full inspiration with partial and slow escape of pent up breath through clenched teeth.

During an attack the child stood on her cot and ceased breathing for from twenty to thirty seconds, then bent her body far over pressing her hands against her abdomen and giving exit to the pent up breath in a series of noisy grunts, terminating in a whining cry. She then breathed noisily and fast until the next attack a few minutes later.

She was isolated from her parents for thirty-six hours at a time and her noisy respiration and grunts could be heard all over the floor on which her room was situated. The attacks seemed independent of emotion; they occurred when the child was apparently quiet and satisfied with her surroundings. She slept scarcely at all and the persistency of her performances was a wonder to all beholders. She was dismissed unimproved, although all reasonable forms of therapy were tried.

Case 4.—J. L., a boy, aged 12 years, was brought to the Clinic, Feb. 24, 1921. In January, 1920, he had had what was diagnosed as influenza, which was characterized by high temperature, malaise and headache, lasting a few weeks. From that time he had slept badly. Five months later, he had developed attacks of breath-holding with noisy respirations. These attacks usually occurred at night and had persisted up to the time of examination.

During an attack the boy usually stood, took a deep breath, held it with his chin depressed on his chest and waved his arms around irregularly. After fifteen seconds he rubbed his abdomen, released his breath, and fell over on his right side. On one occasion ten attacks occurred in five minutes.

During observation of the boy in the hospital for twelve days he was quiet and well behaved. He repeatedly promised to try to control his attacks, but as soon as the promise was given he went off into another series. He slept badly, and the attacks continued night and day. He was dismissed, little improved.

Case 5.—R. J., a boy, aged 10 years, was brought to the Clinic, July 23, 1921. In November, 1920, he had had what was diagnosed as measles, after which he had become restless and fidgety and had slept badly. A few weeks later, he had begun to have respiratory difficulty during which he had attacks of breath-holding. In December, 1920, he had had a general epileptiform convulsion in his sleep. In January, 1921, just after tonsillectomy, his disposition had changed; he had become rebellious, irritable and intolerable, and attacks of breath-holding continued night and day with little rest.

During an attack the boy suddenly jumped from the place on which he was seated and jumped up and down on his toes, passing his hands through his hair, with his breath held in full inspiration. A few seconds later he released his breath, belched a few times, and sat down. His face twitched in a haphazard manner toward the end of the performance.

In the hospital the child was tied down and isolated; nevertheless he went through a modification of his attack with breath-holding. He was noisy and sleepless and intensely irritable. Communications after his dismissal showed that in October, 1921, he had improved and was only having one attack a week.

Case 6.—R. H. T., a boy, aged 6 years, was brought to the Clinic, November, 1921. One night in January, 1920, he had become restless and had slept badly. In the morning he had definite internal strabismus. His temperature had been 100 for a few days, and then the strabismus had disappeared; from then on he had slept badly, and his character had markedly changed. He sang and whistled all night and slept little during the day. A year before he had developed constant noisy breathing and breath-holding attacks. A few months before, he had begun to fall unconscious, rising, however, almost immediately.

The boy was impulsive, noisy, restless and very aggressive. He panted loudly and from time to time held his breath, became cyanosed, and fell unconscious for a fraction of a minute. Moreover, he often fell down when not holding his breath. He had to be strapped in bed to prevent his falling

out, as when unrestrained he invariably stood to breathe noisily and hold his breath, often toppling over at the climax of the performance. He went into bursts of passion for no adequate reason, was abusive, used bad language and attacked the nurses and physicians with feet and fists. In spite of this he was fairly intelligent. When asleep he breathed normally, but he seldom slept. The combination of an excitable, pugilistic manner, gusty, loud breathing and incessant chatter characterized his daily appearance. He was under observation for four months and was dismissed somewhat improved but liable to relapse under emotional influences.

Case 7.—Miss M. G., a music teacher, aged 20 years, came to the Clinic, Nov. 16, 1921, complaining of drowsiness and difficulty in breathing. In February, 1920, she had had what was diagnosed as influenza. She had been ill for six weeks with moderate fever, diplopia, blurred vision and marked lethargy and prostration; this lethargy had persisted so that recently she slept from 8 p. m. until 9 a. m., and often for some hours during the day; she had extreme difficulty in remaining awake. She felt great discomfort when standing erect

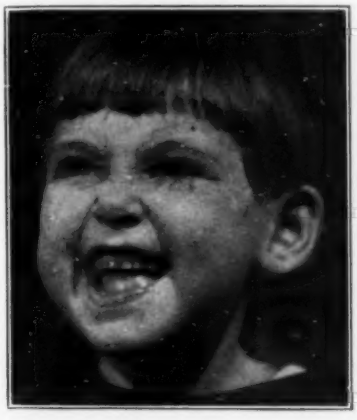


Fig. 3 (Case 8).—General expression of the patient.

or undergoing moderate exertion because of intolerable dyspnea. She spent most of her time recumbent and avoided any exertion. She had gained 20 pounds (9.07 kg.) since her illness.

Respiration and pulse rates varied with sitting, standing and prone positions. When the patient was recumbent and at complete rest, her pulse rate averaged 75 and her respiration 14; when standing or with the slightest exertion, her respirations increased to 30 or 40, her pulse became uncountable, her face became flushed, and all the accessory muscles of respiration, especially the sternocleidomastoids, stood out prominently. There was a parkinsonian appearance in her gait, facies and posture, and she had definite tremor of the hands.

Case 8.—D. F., a child, aged 4 years, was brought to the Clinic, April, 1922. She had had influenza in the spring of 1919, after which she ceased to walk, became backward in development, irritable and noisy, and slept badly. She forgot all speech she had learned; a few months later she began to walk again unsteadily and clumsily. There was little change and no development of mental faculties from the age of 12 months until examination. In March,

1922, she again had a febrile attack associated with cough and malaise. She was ill four days with a temperature of 104 F., and thereafter again ceased to walk. On the last day of her illness she suddenly began to breathe noisily and rapidly and at night had attacks of breath-holding. She began to slobber saliva and developed a habit of constantly sucking three fingers of her right hand.

The child was obviously unintelligent. While observed, without any display of emotion, she puffed away noisily, commencing with a period of apnea and then breathing faster and louder until a climax was reached, after about thirty respirations, when she made a grimace (Fig. 3), held her breath for almost fifteen seconds and made athetoid movements with her hands. She then usually released her breath, waved her arms around aimlessly and panted loudly. This gradually diminished until a period of apnea was reached, when the cycle was again and again repeated for as long as she was under observation. She was dirty in her habits, and unable to walk. Her lower limbs were spastic. She hardly used her left side, and on this side Babinski's sign was positive. Her noisy respirations could be heard at a distance, and these only ceased when she ate and slept. She is still under observation.

GENERAL CONSIDERATIONS

Complete physical, neurologic, and laboratory studies were made in most of the cases, with no more positive results than those mentioned. Attempts to substantiate the suggestion of tetany in a few of the patients by means of blood examination and electrical reactions were unsuccessful. Our first impression of these patients was that their individual performance was a functional disorder of hysterical nature, and the treatment was directed accordingly. As patient after patient showing the same characteristics arrived within a relatively short time, the disease assumed an epidemic character, and we had to seek a different diagnosis. Reviewing the individual histories, we discovered that these patients nearly all dated their illness from the first three months of 1920 and, moreover, some of them had had an initial illness, definitely encephalitic, and some had evidence of gross cerebral damage (Cases 1, 7 and 8). Eventually we had little doubt with regard to the nature of the disease, and its persistency and failure to respond to ordinary therapeutic measures confirmed the diagnosis.

There was a marked resemblance in the paroxysmal disturbances of respiration in the first five patients. Essentially their syndrome consisted of assuming the erect posture, breathing noisily, holding the breath with bodily contortions and releasing the breath in a position best suited for complete expiration.

Cyanosis with partial loss of consciousness, falling, and petit mal-like attacks were common and present in three cases, and all the patients had the marked insomnia and metamorphosis of character so well described by earlier observers.

The only adult of the series (Case 6) had a syndrome observed in no other patient. In many ways her condition resembled that of an

animal with both vagi cut. She had no respiratory reserve to draw on; at rest she was comfortable, but her breathing was always maximal in depth, and exertion could not be tolerated.

Localization of the lesion is a matter of speculation; probably a diffuse process was present. Case 6 was the only one in which one dared to postulate a location of damage as in or around the respiratory center in the medulla.

Endeavor was made to investigate the psychologic mechanism of these patients. Their emotional, rebellious manner was against them, and at times it seemed as if their performances were gone through wilfully, that they themselves could end them if they wished. It seemed that all that was needed was some stern discipline and isolation to produce an amelioration. We soon found out our mistake, as neither of these measures, or any others had the slightest effect. Hypnotism was ineffectual, and direct questioning as to why they went through their movement produced the reply: "Because I want to," or "Because I feel better when I do it."

The lengthy period of the illness, on the average eighteen months, was not more striking than the stereotyped character of the movements which were persisted in day and night with monotonous regularity. The patients were extremely difficult to manage and disturbed all around them.

Two patients, Cases 2 and 8, have been seen recently; they have not improved. The future of the other six patients is as yet unknown; time alone will show whether they will recover and grow into normal adults. The epidemic of encephalitis provided many strange and bizarre pictures, but few surpassed the appearance of these little children at the height of their illness. It is a syndrome that has to be added to the kaleidoscopic picture of epidemic encephalitis.

SUMMARY

1. During the year 1921 eight patients, seven of whom were children, were under observation at the Mayo Clinic, suffering from a disturbance of the respiratory rhythm.

2. While four of the patients had had an infectious illness worthy of the diagnosis of epidemic encephalitis, the remaining patients dated their illnesses from an infectious episode or febrile illness. In seven patients the onset occurred during the first three months of 1920.

3. The main features of the clinical picture in these cases were paroxysmal stretching, breath-holding, grunting and forced noisy respirations. In others there was constant dyspnea associated in one case with posture.

4. The average duration of the illness was seventeen months before examination at the Clinic, and in a few cases there was an appreciable gap between the initial illness and the appearance of the prominent symptoms.

5. The perisistency of the symptoms contrasted with the peculiar nature of the disease which was more manifest by night than by day. It was relatively unaffected by various forms of therapy.

6. Combined with the peculiar paroxysmal respiratory attacks were changes in character. The patients were noisy, disobedient, and passionate, whereas formerly they had been well behaved. There was marked insomnia with inversion of the sleep rhythm in the seven children and abnormal drowsiness in the one adult.

PSYCHOPATHOLOGY AND ORGANIC DISEASE*

SMITH ELY JELLIFFE, A.M., M.D., PH.D.
NEW YORK

About ten years ago I first brought to the attention of this body certain reflections bearing on a relationship between neural integrative factors and what in general was considered as disease. Having become saturated with certain conceptions concerning the importance of feeling and thought factors in human pathology, the translation of Déjerine and of Dubois were only natural expressions of my growing beliefs.

But it soon seemed apparent that the intellectual dialectics of Dubois were not quite fundamental enough; without a corresponding personality makeup behind them the principles are insufficient. Déjerine's emphasis on the feeling—the emotional factors—went nearer to the heart of the problem, but here again the emphasis seemed one-sided. The old faculty psychology, as applied in therapeutics, now dealt with the intellectual functions, now with the emotions, and I even took a shy at the will in my translation of Payot's "Education of the Will." Out of all of this we were brought back to the hippocratic doctrine that the organism, as a whole, must be the object of search and portal of entry made where the organism, as a whole, really lived. It does not live in the intellect, it does not reside in the feelings, it is not at home in the will. None of these "belong." And thus our search for a unity led us into the unconscious—that accumulation of life's experiences (engrammes) which in their zoologic synthesis have been accruing during about a thousand million years.

"What are we in fact," asks Bergson. "What is our character, if not the condensation of the history we have lived from our birth, nay, even before our birth, since we bring with us prenatal dispositions? . . . Doubtless we think with only a small part of our past, but it is with our entire past, including the original bent of our soul that we desire, will, and act. Our past, then, as a whole, is made manifest to us in its impulse; it is felt in the form of tendency, although only a small part of it is known in the form of idea."

Life may then be expressed, if I may take a phrase from our President's masterly address, in the form of a fraction in which the numerator may stand for our conscious idea of things, and the denominator for that past of which we have just spoken, the unconscious. Let me put this in the arithmetical form of a proportion, thus: As the numerator—from minute to minute: is to the demoninator—one thousand million

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* Condensation of paper read at the Forty-Eighth Annual Meeting of the American Neurological Association, Washington, D. C., May, 1922.

years :: so is our conscious idea as to what is happening in life : to the unconscious forces (phyletic memory patterns) that really permit it to happen.

If this is true, our really ever understanding anything looks hopeless. Yet, with that temerity that has ever characterized my efforts here, I venture to bring to your attention, even if summarily, some reflections that may be of assistance in enlarging that numerator, our conscious control of the hidden factors that bring about disease.

It is because of my belief that within the interests that bind this body into a neuropsychiatric society there is to be found the most hopeful outlook for a neohippocratic medicine that I dare to do this.

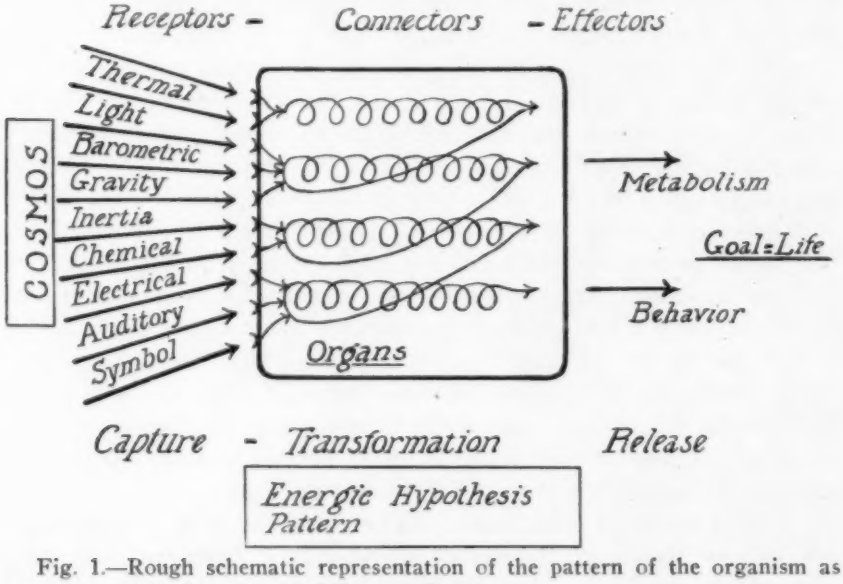

Fig. 1.—Rough schematic representation of the pattern of the organism as a capturer, transformer and deliverer of energy.

It may become possible thereby to understand some of the activities of the organism as a whole.

Permit me to give a rough scheme of this (Fig. 1). The organism, as a whole, carries out its energic program of capturing, transforming and delivering energy. The cosmos is its petrol tank; the organs, its structuralized functional transformers. These are integrated and coordinated to act, as a whole, through its nervous mechanisms, vegetative, sensorimotor, symbolic, to deliver itself in its *metabolic upkeep* and its *behavior*. The goal is the continuance of life; immortality.

EPOCHAL PERIODS THROUGH WHICH ORGANISM PASSES

Also permit me as sketchy a scheme as to the major epochal periods through which each organism passes, recapitulating the history of its

prenatal past as well as its individual participation in that experience which has been molding it throughout its geologic time period. That thousand million years of recapitulation hurries by in the nine months of intrauterine life.

Archaic Period.—Dr. Tilney's time consuming and masterly studies of the developmental history of the nervous system give us an opportunity to peer within some of the things which are so rapidly forming in this period, to which the term archaic may be applied.

In the comparatively insignificant period of nine months, one thousand million years is traversed. From primordial ooze to man, in one majestic sweep, the creative impulse shows its handiwork. If one wishes to call it God, it is only a matter of terminology. No name will ever be adequate to encompass the facts. In the short period I have for this presentation, I cannot commence to touch on the implications which surround the formulation here sketched. So far as human

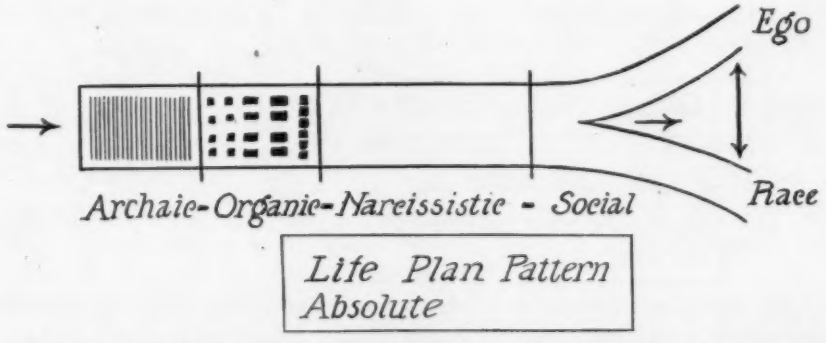

Fig. 2.—Rough schematic representation of the life plan pattern in its phyletic dynamic sense, with division of periods, archaic, organic, narcisistic and social.

pathology is concerned, all that I wish to emphasize at this time is that defects of development in this period are accessible to the newer psychopathologic technics and are integrated into the hypotheses which I would present to you. Man has retained his phyletic theomorphic capacities in the soma. It is here maintained that they are capable of projection into the symbolic sphere in the highest of known animals, man, and if recognizable, may be of fundamental service in the problems of pathology. When our concepts become big enough the material is at hand to be interpreted.

Organic Period.—A further glance at our second diagram shows the next period of development is the organic. By this is meant that man, having been born, commences to use his tools. Individual experiences become more striking and acute, and an important period of organ rivalry sets in. The need of oxygen brings distress; the reflex act is the

cry; this starts the respiratory rhythm. I need not discuss the complicated physiologic hypotheses. Here in essence is to be found the need-oxygen (physicochemical level), the organic act, respiration (sensorimotor level), and the cry (the symbolic level). Crying satisfied a need; it is now used to attempt to gratify all needs. But with the act of nutrition, there occurs to use the vernacular, the first "fifty-fifty" in the child's life. It cannot "holler and swaller" at the same time. Choice must be exercised, inhibition becomes operative—again a full discussion of the problems would occupy the day. In fine, repression has won, in part or completely, and healthy adaptation has taken place. This rivalry goes on among all the organs and has an enormously intricate and subtle history, as will later be discussed in our outline of the mechanisms by which the supremacy of the genital zone craving becomes established. Many problems of so-called constitutional disease, often thought of as congenital or inherited, may be reexamined to

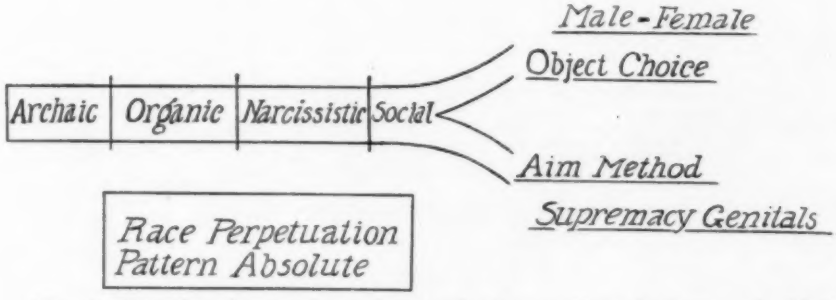

Fig. 3.—Rough scheme of psychosexual stage pattern in terms of object choice and in supremacy of genital zone.

advantage from this standpoint of libido rivalry. The individual's psychologic level mechanism may be of paramount importance in determining structural alterations in this infantile period of adjustment. Here Pavloff's general thesis of the *conditioned reflex* is a conceptual tool of great value in comprehending the symbolic identifications.[1]

Narcisistic Period.—A third and a fourth definition are to be attached to Figure 2. The third period of development posited is the "narcisistic." The individual has become an integrated personality. He passes out of the period of "cylinder adjustments," to his "place in the road." The inner machine now passes to the conception of *my* car: I, John Jones. His cravings are still segmented so far as their somatic activities are concerned. But they are not so radically felt as such. Narcissus has been born. Self love, reflex activities conditioned on the basis of purely egoistic strivings, libido attachments to mirror

1. Freud: Three Contributions to the Theory of Sex, Nerv. & Ment. Dis., Monograph Series 7.

pictures of the self in all of its developmental stages, these are the earmarks of this period. Permit me to emphasize the fact that I am talking now in terms of the denominator, of the phyletic memory patterns, not in terms of the conscious. We are here interested in learning what narcisism is, in the terms of unconscious attachment, and we shall see later that it, too, offers complicated and subtle differentiations.

The Socialized Individual.—Finally, man advances into the territory of a socialized individual. He has left the self, and the values of social integration commence to be those of permanent value. No matter how complexly discussed may be those values which roughly speaking are here termed social, it may be seen that when the individual is subjected to the divesting process of the psychanalytic technic, these goals of his striving stand out in naked relief to his numerator, his consciously adopted, usually quite flattering, estimate of his motives. It is here that the acid test of the psychanalytic technic cuts deep into reality, and one may see almost at a glance the coordination between the individual's various segmental strivings and the stage of psychosexual expression attained.

If life's chief goal is its continuation, then in the phyletic sense, waving aside all the petty conscious notions of what is meant by sex, it may be seen that the urge for continuance has fashioned itself into every structure of the body. And every cell of living matter exists only to carry on the supreme work of creation. That is its fate, if one wishes so to regard it; its promise as well.

Adult, that is, socialized, psychosexual evolution, is, then, the highest goal that man can reach, and falling short of this, his machine lags behind either as a receiver, a transformer or a deliverer of energy. Here again conscious rationalizations as to what constitutes adult psychosexual evolution must be put aside in an application of psychopathologic data to the study of disease and human suffering. In their time and place such ethical systems have had almost sublime values, but like many a goodly apple, rotten at the core, the individuals professing them may be deceiving both themselves as well as others, and the ethical systems themselves have been utilized in a sense negative to their fundamental phyletic values.

In Freud's masterly study, "Three Contributions to the Theory of Sex," we see that, phyletically speaking, object choice and supremacy of the genital zones must be coordinated into a socialized sexual pattern. The object choice pattern has been built up on a heterosexual foundation for millions of years; likewise the germ plasm structuralizations have demanded that they be put to socially constructive utilizations, else the individual, be it lowly plant or highly evolved man, will be thrust aside in the relentless march of progressive,

emergent evolution. The psychanalytic theory maintains that these instinctive patterns are capable of analysis within the individual. Behaviorism, as a fragment of realistic science, it welcomes, but it holds that this highly useful observational science can be aided by intellectual tools through the utilizations of the psychanalytic technic.

Let us turn to our next diagram, Figure 4, and see, roughly outlined, what this technic offers for the comprehension of the instinctive mechanisms surrounding object choice. This has been envisaged by Freud as the Oedipus complex, or Oedipus hypothesis, by the utilization

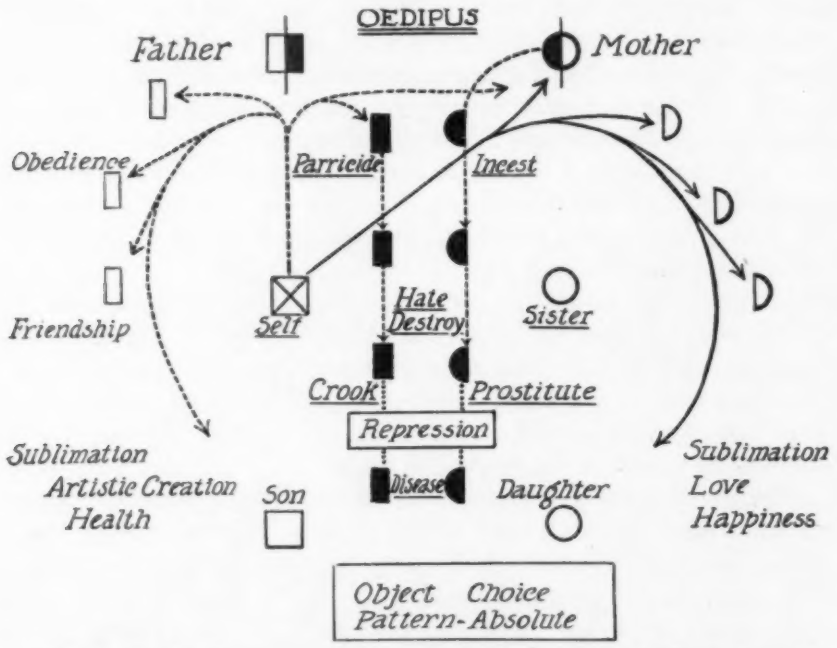

Fig. 4.—Crude diagrammatic representation of the mechanisms by which the adult psychosexual object is reached in terms of the Oedipus hypothesis. The psychologic splitting of the mother-father image is roughly indicated.

of which the unconscious symbolizations throw light on the stage of psychosexual evolution of the individual under consideration. It cannot be too strongly urged that we are dealing with analytic, i. e., individual problems. Their synthetic aspect, i. e., the doctrinal generalizations, must be left outside for the time being.

The diagram is sufficiently explicit, but I cannot forego the observation that this diagram must be read not in the sense of a purely conscious series of behavioristic reactions, but as a representation that must be conceptualized from the standpoint of the denominator, that is, the unconscious in the psychanalytic theory.

Almost all of the misconstruing comments on the Oedipus complex are due to the failure to comprehend this. When in chemical symbols we say that 5 H_2O + 6 CO_2 [+ solar energy + chlorophyl] = $C_6H_{10}O_5$ + 6 O_2; that is, water and carbon dioxid in the presence of solar energy (sunshine) and through the catalytic action of chlorophyl, yield through a series of reactions, starch, oxygen, etc., the nonchemically trained observer is incapable of understanding this simple symbolic statement of vital processes going on in the chlorophyl-bearing leaves of plants. The Oedipus symbolic statement is equally outside the ken of the non-

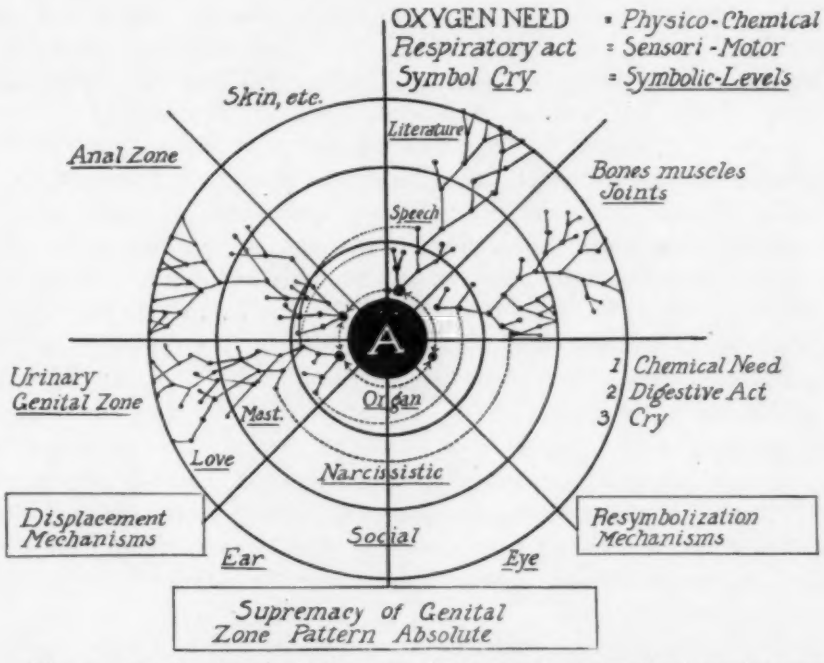

Fig. 5.—Rough scheme of stages in development of socialized psychosexual aim pattern. In this diagram the linear form, as in Figures 2 and 3 is changed. The various stages *A*, archaic, organic, narcisistic and social are represented in widening circles of resymbolized patterns of activity each showing the delivery mechanisms working at more adult levels. Displacement is also represented. The socialized end products which constitute behavior in its various aspects cannot be put in the diagram, but they can be readily conceptualized. Each segment stands for a segmental craving structuralization (schematic).

analytically practiced mind. Hence, most of the footless discussions about the Oedipus hypothesis and its implications, not the least of which was that of Mills at our last meeting.

The rough diagram in Figure 5 attempts to indicate a complex series of mechanisms. If it be conceived in a dynamic sense, it would portray the various physiologic activities of the human body. In terms of

Figures 1 and 2 these are all carrying on (*a*) the function of self preservation (metabolism) and of (*b*) race perpetuation (social behavior) in its various aspects. It has already been indicated how the displacement mechanism under inhibition, or repression, can push energy into this or that channel. With advancing development the simple manifestations of the delivery systems become more and more complex, preparing the organs, the Narcissus, and finally the socialized adult to deliver his energy at more fully integrated and coordinated ethically valuable levels. Thus, out at the periphery of this diagram can be shown how energy deliverable through an organic satisfaction at a more primitive level may be displaced to another area and then resymbolized and gain satisfactory, and hence health giving, expression through another group of organs, at a higher, that is, a more socialized level. When, so to speak, it is said, "One whistles to keep up his courage," the craving to run away, by means of the leg muscles, is satisfied through the whistling. This simple illustration may be amplified a thousand fold, and the Freudian mechanisms of condensation, secondary elaboration, displacement, conversion, substitution, projection, etc., are keenly thought out formulas through which the dynamically thinking observer can come to understand human behavior, either at its metabolic, its sensorimotor or its symbolic level. The organism works as a whole. Physics and chemistry, sensation and motion, thinking and feeling, are all operating parts. Should we seek to grasp the largest of the integrating formulas, that is, a true integral calculus applicable to human behavior, symbolically expressed, such calculus formulas, differential as well as integral, it is here submitted, can be partially glimpsed in the schemes here sketched, when viewed more from the denominator standpoint, quadmillions to one, by means of the psychanalytic technic.

REPORT OF A CASE

I shall present my case in tabloid form. I present the bare outline of a case (Fig. 6), which, permit me to elaborate somewhat.

In 1914, an old friend came to my office and dramatically said, "For God's sake, save my wife! The doctors have given her six months to live, and I am out of my mind." In response to my questions he said "She had kidney trouble, a blood pressure of 240! and according to the doctors, unless she dieted, went to bed, did this and did not do that, she would die in six months."

I assured him that it could not be as bad as he said. He was upset and his fears had magnified what had been told him. I said that I was sorry but did not specialize in kidney disease, etc. His attendant and consultant physicians were the best in the city; in fact, they had national repute and could be relied on. I knew his wife was of a very

active type, but I said it would be better for her if she slowed down a bit and possibly gave up her plan of stumping the state for woman's suffrage, or what not.

He insisted on my doing something. So I told him I would send her to one of the city's best hospitals, to one of the best internists, and get as complete a balance sheet of her condition as I could get, and then, as he was an engineer, I would translate, as best I could, the Greek and Latin terminology of the medical lingo, into mechanical terms with which he was familiar.

So I did. She was examined by all the methods known to internists' lore of the time. She was under observation for two or three weeks. The problems involved were, clinically speaking, quite banal. There was a cardiovascular-renal syndrome known to all medical men, and

A woman, aged 36, married, children ♀, ♂, ♀, ♀, nephritis-hypertension.

Symptoms Headache four years. Blood pressure 240-250 mm. Albumin. Diminished urea output. Retention. Asthenia. Edema. Dyspnea. Constipation +++. Slight momentary lapses.

Behavior Able, energetic, cultivated interests in home, children, society. Two girls in family. Devoted father; beautiful, much admired mother. Large family group of professional people. Never peculiar. No eccentricities.

Unconscious *Oedipus evolutions*, defective. Strong father fixation. Rejection of male. Homophilic. *Supremacy of genital zone*, defective. Urinary fixations. Strong anal erotic components.

Fig. 6.—Outline of Case.

hypertension of from 220 to 240 mm. systolic pressure, with nephritis. The treatment prescribed was rest in bed, plenty of water, a restricted diet, plenty of restrictions. The prognosis was dubious and serious; she might live for some time if she followed the treatment indicated; if not she would probably die within a short time.

I asked concerning the cause of the nephritis. The physician said it was the high tension. I said, "Why the high tension?" "The nephritis," he answered. "And where do we get off this circle?" "We don't," he asserted. And there we were. Of course, I am abbreviating the conversation. A review of the world's literature during the past four or five years on this problem shows that this is its present status. As Widal, in answer to a similar inquiry I made to him last summer, at the close of a brilliant bedside clinic on almost a facsimile of my patient, said, "C'est les mysteres! Internal medicine, so far as it has

gone, cannot break into the circle, and looks, when it looks at all, at the 'mysteries.'"

When I explained to my engineer friend what I had been told, he saw the difficulty, but was surprised at the static position in which the internists left the case. I agreed with him. After much discussion I finally consented to make a tentative appraisal of the intrapsychic situation and try to determine whether, in terms of what has here been formulated, there was any look in on the dynamic side.

Within two weeks it was quite apparent that the unconscious material afforded illuminating glimpses of some of the "mysteries," and told us *why the organism as a whole* was not functioning satisfactorily, although I was not able in that time to determine why the cardiovascular-renal components were the structures which showed the most evident signs of breakdown (that is, from present day clinical criteria).

I then proposed to conduct a research. If, in the crude thought of centuries, the mind was said to influence the body, what could the refinements of analytic technic show as to such influences? Or, since modern psychopathology rejects this setting off of opposites, body versus mind, what could such a technic show as to the mental, that is, the symbolic level activities, going on in the individual? These the internist knows little about, except as he mouths such vague monstrosities as "nervous," or "emotional" or "psychic." The internist is, for the most part, working at the physicochemical level. Like Yank, in O'Neil's "Hairy Ape," he thinks he "belongs" because he stokes the fires. He is iron and steel. Our present internist's conception of the "human machine" is as crude as Yank's conception of the world which broke him. I am not unmindful of the legitimate protest against such a statement, as evidenced, for example, in Kraus' "Allgemeine Pathologie der Person," in which, fortunately, it may be seen that internal medicine is breaking away from the static molds of descriptive science which have been building up too onesidedly for the past fifty years. Nevertheless, I maintain that when the time comes that Claude Bernard dreamed of—"when the physiologist, the philosopher, and the poet would talk the same language, and understand each other"—a true science of medicine will be possible. That time has not yet arrived. We have a few physiologists, but where are the medical philosophers? As for seers in medicine, they are too few and are mostly despised and rejected of men. I need only refer to one medical poet, philosopher, and physiologist, who after thirty years of contumely and most prejudiced criticism, is finally recognized as a genius, and whose illumined hypotheses are making it possible to understand the enormous rôle that psychopathology plays, not only in the neuroses and psychoses, but in what is termed constitutional disease. But to return to our patient:

I cannot recapitulate all the evidence, but the very first finding in the "unconscious" contained some interesting material. While in the hospital she had dreamed the following which was a seminightmare:

"There was a road along which two men were driving like mad in single-horse racing sulkies. As they went by in a cloud of dust, a woman with disheavelled hair came from a house facing the road wringing her hands and screaming at the top of her voice. The men and horses went up a steep hill, and as one reached the top he turned sharply to the left and ran into a stone wall about two feet high and smashed the sulky all to pieces."

Now I know to the nonanalytically trained observer this means as little as the sight of a small red stained rod under the microscope means to one who does not know anything about the tubercle bacillus.

To the patient I said, "What about it?"

"About what," she said?

"Well imagine yourself one of those riders."

"I'd be crazy," she said.

Well, I said, "It looks as if somebody was destroying something, at all events, and maybe the *wish* to destroy has something to do with your own breakdown." "Let us go further." Then her free associations were obtained, and for several sessions we worked on this dream.

To give the results of these investigations in extenso would be as wearisome as to give in minute detail all of the intricate directions for carrying out a Wassermann test. The general findings showed an obvious difficulty in her object choice. According to the principles of the Oedipus hypothesis, she showed a strong unconscious father fixation. He was the man of the sulky. A part of him, the one that ran into the stone wall and smashed up the machine, was related to the incestuous component of an infantile fixation period; but to the student of unconscious processes as modified through the dream work, and as further extended by the technic of free associations, an enormous amount of material may be recorded, just as the physician who finds the tubercle bacillus in a patient's sputum has opened up an enormous amount of material heretofore unknown (Fig. 4).

Here, then, one finds the first obvious failure in the energy delivery system, since it had become fixed (conditioned) for infantile rather than for adult psychosexual functioning. The psychopathologist can envisage some high voltage energy seeking an adequate pathway for discharge (*racing horses on the road*), and not finding the adequate outlet, backfires and creates havoc somewhere in the machine. This is the general rough idea. Can an analytic Franklin conceive, with his kite and string, how to determine the line of discharge? That is, can the analytic technic show why the cardiovascular-renal structures were chosen as the lightning rod for grounding this faultily delivered

energy? If so, then, in general, we have the leading features of our problem laid bare: *faulty psychosexual evolution* in terms of *object choice,* and *supremacy of the genital zones.*

Every psychopathologist working with the analytic technic knows that the stage of the Oedipus formula turns up fairly early in an analysis. It may be that Freud's genius in pointing out the way enables us to recognize the main indicia of difficulties in its development. The traveler from New York to San Francisco recognizes Buffalo, Chicago, St. Paul, Denver, Salt Lake City as stopping places on the road; so the analyst can observe the symbolizations of the *Archaic, Organic Narcisistic* and *Social* stages in the growth of the individual to adult psychosexual development. But there are innumerable stations between these larger more or less arbitrarily named stopping places. The Libido is, however, ever on the go. It stops nowhere. It is the insufficiency of the intellect that creates the need of static stopping places, of giving dead names to dynamic processes.

Psychopathology is still working to analyze the displacements, the side tracking, splittings, and condensations of energy traversing the somatic segmental pathways in the evolutionary urge toward a truly creative use of the segmental structures.

Judging from conscious criteria, the fact that our patient had four children all growing up and free from gross defect would argue that the supremacy of the genital zone had reached an adult stage in the plan of psychosexual evolution. This is the general intellectual conception. But on looking over the accumulating dream material, a sufficient number of reasons are found which tend to explain why the patient was frigid in her intercourse with her husband, even frigid to manual or any type of contact with the genitals. The evidence was plain that a fixation had taken place even before the clitoris could serve as a guide to the zone supremacy. There was plainly indicated in the dream material that vesical and urethral fixations were conditioned and that no supremacy had been reached beyond this stage.

Marked constipation which had resisted over twenty years of attempted treatment by scores of procedures was soon traced to its anal erotic sources—another libido displacement—and was effectively relieved in a few months. One bit of dream evidence bearing on the anal eroticism is worthy of record. After I had known the patient about eighteen months and she had been free from constipation for over a year, she had a short period of relapse. She came in one day and laughingly asked me what I thought of this dream:

She and B. (her maid) were trying to smuggle a couple of boxes filled with long bottles into a small closet on the second floor. It was locked from the inside and she had to descend to the cellar and ascend a circular staircase in order to unfasten this closet door. As she

started going up the circular staircase she noticed a Chinese mask on the wall of the cellar.

"The bottles?" I asked. "Pluto. Isn't the dream a cute one? It looks as if my attack of constipation was hankering for some gratification."

"But why the Chinese mask and the circular staircase"? This was addressed to me.

The Chinese mask was soon resolved as Father. The circular staircase, her intestinal tract. Further analysis resolved the outlines of the pederastic aspect of the anal erotic wish. Whereas it seems a far cry from Fabre's story of the impregnated spider who immediately devours her mate for food for the offspring, it is by no means an unrecognized factor in certain human matings that the "bringing home the bacon" for the sake of support of self and children (often the latter bring but narcisistic replicas of the self) is, if not the chief motive, certainly near consciousness. In this connection the myth of Lot's wife and the story of Sodom and Gomorrah would repay reading in the light of unconscious fixations.[2]

The temporary constipation regression cleared up, and for seven years now there has been no necessity for treatment for constipation. The constipation, that is, the anal erotic fixations also showed on analysis much concealed sadistic material directed toward the homosexual, much envied mother (unconscious), displaced and concealed behind the heterosexual, husband-father image. This mother rivalry also was marked (unconscious) and hidden behind urinary phantasies. In childhood water plays were adored. They were numerous and were followed with fascinated excitement; copious, almost abnormal, water drinking afforded greater somatic outlet as well. The urinary gratifications were all of this intense quality. They thus demanded a large renal output, and they got it. In a complicated and subtle manner from the ages of 3, 4 and 5 years, the unconscious urinary phantasies made use of the cardiovascular-renal mechanism to gratify an almost feverish urge to overcome the mother and later the mother imago (homosexual) substitutes. Hence the constant narcisistic homosexual unconscious symbolizations which thoroughout the entire analysis were persistent sign-posts of the retardation in complete psychosexual development, both as to object choice and to supremacy of the genital zones.

It would take many more hours to present the complete analysis, but I hope I have given a glimpse of the problems as seen from the analytic standpoint.[3]

64 West Fifty-Sixth Street.

2. Compare Jones: The Symbolism of Salt, in the Unconscious, Collected papers, Ed. 2, New York, William Wood & Co., 1921.

3. Jelliffe, S. E.: Paleopsychology. A Tentative Sketch of the Evolution of Symbolic Function, Psychoanalytic Rev., to be published, January, 1923.

POSTENCEPHALITIC DEFORMITIES OF MOTION

A LECTURE ILLUSTRATED BY MOTION PICTURES*

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Two groups of cases each on a separate reel, altogether about 2,000 feet of film, are shown. The first group includes cases showing deformities of motion and representing types of cases with recognized syndromes, that is to say, nosologic entities. After pointing out the varieties of motility defect, I will show the second reel illustrating unusual types of postencephalitic deformities of motion. In all of the cases studied, there are adventitious elements of motion resulting in definite deformities of movement.

A comparative study of these two groups of cases shows that the elements of motor deformity in each are analogous and in some instances identical. Though the histopathology doubtless differs, the localization of lesions must be the same. Indeed it suggests a definite relationship perhaps even between the underlying pathologic etiology.

In the two series of cases we see the motility disorders affecting the static, the kinetic, the synergistic mechanisms and their harmonious activities.

REEL 1.—The initial presentation in this reel shows a group of cases illustrating various types of dystonia musculorum deformans. The first three patients are all members of the same family, two sisters and a brother. The least afflicted of the three was the brother. The motor disturbances began in his thirteenth year affecting at first the finer movements of the right hand. The right leg, including the foot, then showed spasmodic uncontrollable involuntary attitudes in attempts at movement. The sudden flexion at the hip and knee and inward rotation at the hip produced the characteristic attitudes. The peculiar overflow of kinetic energy with the tendency to postural fixation is represented in its incipiency in this case, and the progress of the disease as the kinetic and static mechanism becomes more involved is demonstrated in the more severely afflicted sisters. In these two young women the static mechanism has assumed such control that we have definite postural defects. The associated action of the two systems is defective, producing athetoid movement of the upper extremities in the case of the sister most severely affected.

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* Read at the Forty-Eighth Annual Meeting of the American Neurological Association, Washington, D. C., May, 1922.

* Patients from the Neurological Service of the Montefiore Hospital.

The next case of dystonia shows extreme sustained spasms—kinetic overflow and tendency to temporary postural fixation. The first symptom of the disease showed when the patient was 12 years old and began as a stiffening of the right leg with a resulting rather awkward gait. The picture shows a voluminous musculature. There are present characteristic though only temporary attitudes and poses, but there is a mobile type of spasm with kinetic overflow without static fixation, showing that the static mechanism giving rise to sustained postures is least affected. The muscular group involvement is much more general than in the previous patients.

The next case of dystonia is that of a girl of 17. She presents the so-called dromedary attitude with what has been described as the semilunar foot. The first symptoms appeared in her seventh year as involuntary movements limited to the right foot, spreading gradually to the right hand and arm, then becoming general. This is a case in which the static mechanism was more especially involved so that segmental fixation has given rise to marked deformities. There also appeared sudden ticlike movements of muscle groups of the upper extremities and those of the neck and upper portion of the trunk. These movements have become bizarre and represent almost every type of abnormal motor expression. There are the myoclonia, the tic, the choreiform, mobile spasm, etc., all showing in one patient. Yet with all there are present the distinctive features of both the kinetic and the static types of dystonia. (Drs. I. S. Wechsler and S. Brock [1] introduce an entirely new conception of the several types of dystonia musculorum deformans.)

The dystonia elements in the next case are confined to the upper extremities and the trunk. There is sustained mobile spasm in the sternocleidomastoid and the trapezius muscles of one side with a fixation of the trunk by marked lateral curvature due to the special involvement of the static mechanism (Wechsler and Brock).

This boy of 12 years again presents the mobile spasm type, the kinetic overflow giving rise to constant arrhythmic oscillations of all parts of the body during rest, intensified when effort at voluntary motion is made.

This middle-aged man shows the typical symptoms of progressive degenerative chorea. The hereditary element is absent, but the mental and physical signs began in his forty-fifth year. The features of the case are ticlike movements, involving especially the abdominal muscles, and are identical clinically with the myotonic movements of encephalitis.

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1. Wechsler, I. S., and Brock, S.: Dystonia Musculorum Deformans with Especial Reference to a Myostatic Form and the Occurrence of Decerebrate Rigidity Phenomena, Arch. Neurol. & Psychiat. **8**:538 (Nov.) 1922.

The two cases which I would describe as types of dystonia lenticularis, or better perhaps striatal syndromes, show analogous deformities of motion. The distinctive characters of these movements are discussed in a paper by Dr. J. Ramsay Hunt. Hunt has called attention to the cerebellar elements involved in the pathologic process and in his present paper will discuss the striatal elements involved. These two cases were considered by him in his discussion of a type which he originally described as a progressive cerebellar dyssynergia. The clinical features of the cases consist of deformities of motion suggesting the dyssynergia of multiple sclerosis with tremor-like movements somewhat analogous to paralysis agitans. The one patient, a woman of about 60 years of age, was under observation at the Montefiore Hospital for a long period of years. At her death necropsy showed a fetal adenoma of the thyroid gland with hypertrophy of the gland itself; a large hobnailed liver of the Laennec type and bilateral cystic degenerative changes in the putamen of the lenticular nucleus; also degenerative changes in the vestibular nuclei with softenings in the bulb and cerebellum.

The case of double athetosis shows the universal involvement of the entire skeletal musculature and those of phonation are also involved. The condition is one of congenital origin.

All of those cases have their striking analogues as deformities of motion in those that will be presented in the next reel, and it is for that reason that this first series has been demonstrated.

Reel 2.—The first of the series shows a young girl whose attack of what appears to be a typical chorea came on about three weeks following an acute encephalitis. The movements involving the entire body musculature are, however, a bit more rapid than the usual chorea, and show a tendency to symbolic attitudes indicative of emotion. There is something of a posing and attitudinizing which we sometimes find in cases of chorea. The two latter features, so impressive in certain well recognized organic diseases, suggest a close anatomic relation of these symbolic movements to the basal ganglions of the forebrain. The thalamus and the corpus striatum, which are functionally associated with sensorimotor complexes, serve as the centers especially controlling automatic associated acts and attitudes expressing the fundamental emotions (Goodhart and Tilney). If this case were studied by slow motion photography (bradykinetic analysis), these features would doubtless be brought out.

The case next presented on the screen shows a variety of abnormal movements. Combined with athetoid movements of the upper and lower extremities are ticlike motor expressions of the left sternocleidomastoid with rhythmical oscillations of the head and the trunk. This combination of deformities of motion presents a clinical picture unlike

that seen in any other form of disease. The clinical picture followed within a few months after an acute attack of the infection. As the patient stands supported the movements suggest the striatal type seen in the previous reel and described as dystonia lenticularis.

The following case possesses features of especial interest. The motor syndrome followed about a year after the patient had had an acute attack of encephalitis. The first pictures show her as a young woman apparently in the early twenties. Her face is full of expression, and there is no absence of normal associative movements. There is nothing to suggest a parkinsonian attitude. There is, however, a ticlike movement of the left arm and leg with turning of the head to the right. The movement of the extremities is one of adduction accompanied by a sigh or inspiratory movement. It is distinctly of a unilateral, hemitype. I think it is safe to say that this movement has been seen in no other disease. It suggests the oculo-encephalogyric form, and in a way portrays the agonal, slightly convulsive reaction not infrequently observed just preceding dissolution. It is likewise somewhat analogous to myoclonia; its localization is baffling. I, personally, feel that the pathologic condition lies in the striatum. The left facial paresis is a residuum of the acute encephalitis. As we observe this girl about a year later, the change in her appearance is striking. The abnormal involuntary movement of the head, left arm and thigh has disappeared. Her attitude and posture approach the parkinsonian type. Her face, expression and general appearance suggest maturity—she appears at least ten years older. Another interesting feature is the absence of any rigidity though the familiar posture and attitude are present. It is evident that the latter features giving characteristic design to the pose of paralysis agitans is independent of muscle rigidity. Only later is the latter feature added—the design receives its permanent molding, so to speak.

The middle-aged man next presented demonstrates the champing movements of postencephalitis. The pterygoids are bilaterally involved. The constant chewing movements, here as in other deformities of motion in this disease, cease only during sleep. The patient appears to be ceaselessly chewing.

The next two cases are shown because the parkinsonian design is confined to the typical change of expression of the face, with tremors of the facial muscles and tongue. These common postencephalitic symptoms appeared shortly after the acute illness. Both patients are middle-aged.

In addition to the parkinsonian syndrome, the case next presented shows involvement of the thalamus. Outbursts of spontaneous laughter, uncontrolled, and without attending psychic effect, characterize the illness of this patient.

The young girl with this marked hemiplegia, with the dystonic elements in posture and movement, with the spinal cord involvement as evident by the extensive decubitus, with the cranial nerve symptoms, with parkinsonian tremor, is another encephalitic patient again showing this variety of defect in the kinetic and static mechanisms. The distinctive feature of the case, however, is the peculiar ticlike movements of the left lower extremity. This is induced by lightly touching the extremity at any part. The entire condition may be said to be a residuum of the acute encephalitic process.

This little girl of 7 presents the paralysis agitans facial expression with the rhythmical oscillations of the upper extremities; the lower extremities show a tremor; the latter, however, is of entirely different character and tempo. The lower extremities displaying rigidity, with contracture as well, show no pyramidal tract symptoms, although a spurious ankle clonus can be elicited. When this child is suspended the extremities assume a position of that seen in decerebrate rigidity. A remarkable feature of this case is the spasmodic turning of the head and the eyes strongly to the left. This oculo-encephalogyric movement occasionally persists for several hours. The right sternocleidomastoid in these attacks stands out with remarkable prominence; the mass of muscle has the resistance and the feel of metal. Efforts to overcome the resistance by turning the head to the right increase the already terrific contraction. The unusual posture of the two arms is also strikingly shown associated with the oculo-encephalogyric movement, and is a result of sudden extreme extension of the left arm with strong flexion of the elbow and adduction of the right arm. A study of the remarkable motor phenomena of this case suggests an entirely new conception of associated movements.

That the classical dyssynergic manifestations that characterize multiple sclerosis may have an encephalitic pathology is demonstrated by this young woman. The so-called intention tremor with the muscles of phonation also involved finds its most extreme expression in the wild excursions on any attempt at movement. The disorganization of motor control followed closely on the acute attack of encephalitis.

I am indebted to Dr. Israel Strauss, from whose service at Mt. Sinai Hospital this case was studied and the moving pictures taken. It presents an acute case of encephalitis showing the characteristic myoclonic abdominal movements. Of far greater interest, however, and very unusual are the abductor external rotation movements of the lower part of the leg and foot. They are distinctly of ticlike character and are such as would be expected on the application of an electric current.

The next case is that of a young woman whom I saw in private practice, and the pictures were taken during the attack of acute encephalitis. You will observe a series of myoclonic movements of the muscles

of the quadriceps extensor groups. The movements appear like massive fibrillary tremors. To me they suggest stimulation of groups of anterior horn cells, and from the appearance of this picture of motor activity, I feel that we are dealing with stimulation or irritation, toxic or otherwise, of anterior horn cells of the cord. The same massive fibrillation is seen in the hamstrings and glutei when the patient lies on the abdomen. It is of clinical interest to note that unlike many of the myoclonic movements these were attended by intense pain preceding and during the motor manifestations.

This boy of 12, now in the subacute stage of encephalitis, presents the classic syndrome in every detail of paralysis agitans. During the examination and cinematographic production he fell asleep. Loss of automatic associated movement has resulted in lateral, retropulsion and propulsion, posture, attitude, gait, etc., rarely so complete in a juvenile case of Parkinson's disease.

This little girl of 12 shows bilateral involvement of the pectorals and adductors of the thigh and of the sternocleidomastoid and trapezii muscles. These groups are in bilateral, symmetrical myoclonic movement. The motor phenomena of this case are of a character one may call unique and not seen in any other disease of the nervous system.

The underlying pathology and more especially the localization of the motor manifestations of encephalitis present problems and open an entirely new vista in the realm of deformities of motion.

DISCUSSION

Dr. J. Ramsay Hunt, New York: I should like to say a word about my two cases of striocerebellar tremor which were shown and which will be presented more in detail later.

Some years ago, under the heading of progressive cerebellar tremor, I described a group of cases, the essential symptom of which was a progressive cerebellar tremor. The tremor was sometimes extreme, more so than one usually encounters in such cases. Since my original presentation, which was purely clinical, I have had the opportunity of making studies of the pathology in two cases. In one of these there was primary atrophy of the efferent dentate system of the cerebellum (the dentate nucleus and superior cerebellar peduncles). This type represents a pure cerebellar form of tremor.

In the other case there were symptoms of progressive dyssynergia (cerebellar tremor) associated with coarse rhythmical tremors, which were sometimes spontaneous and occurring at rest. Study of the pathology of this case revealed the lesions of pseudosclerosis, localized in the corpus striatum, the cerebellum and the brain stem. This case also presented the typical lobular cirrhosis of the liver and is, therefore, allied to the group of cases which has been described as pseudosclerosis, progressive lenticular degeneration and dystonia lenticularis. It constitutes a pure tremor type of hepatocerebral degeneration. The tremor in this case is peculiar in that it presents a combination form of the striatal and cerebellar types of tremor, which I would term striocerebellar tremor, the explanation for which I believe is to be found in the simultaneous involvement of the striatal and cerebellar mechanisms.

The second case which Dr. Goodhart has shown is also an example of striocerebellar tremor, presenting distinct evidences of this combined form of tremor. There is the cerebellar tremor of the intention type and in addition spontaneous rhythmical tremor manifestations such as occur in paralysis agitans and Wilson's disease.

Dr. Foster Kennedy, New York: The teaching that has been generally accepted regarding the etiology of tic might well be reconsidered in view of the fact that identical appearing movements are seen as the result of specific midbrain lesions. We have been taught and have accepted the idea that spasmodic tics are invariably of psychogenetic origin. It would seem proper to suggest that these are more probably in the nature of localized release phenomena than the result of a purposive movement, the object of which has been forgotten or lost.

Dr. Charles K. Mills, Philadelphia: I was struck by the fact that in one of Dr. Goodhart's cases double lesions were present, one in the putamen and the other in the cerebellum. I believe when Dr. Hunt first recorded the case it was in a paper entitled "Dyssynergia Cerebellaris Progressiva." It is a question whether the name which Dr. Hunt used was not a misnomer. It gave me the impression that the syndrome of the disease which he was describing was due to a purely cerebellar lesion. To use a striocerebellar explanation may help out a little.

I have one case particularly in my mind, which seemed to me at the time of the reading of Dr. Hunt's paper to be very similar to his so-called dyssynergia cerebellaris. This patient was seen by members of the Philadelphia General Hospital's neurologic staff on various occasions, and I believe that the case was not one of true cerebellar tremor. It is almost impossible in such cases to make any real study of such synergic phenomena as adiadokokinesis and dysmetria.

It is interesting in discussing these questions of movement to remember that the motor symptoms presented are often release phenomena and not due to the direct effects of lesions in the organs affected.

The striatum for instance may be markedly diseased, but the active disorder of movement may be due to the fact that the cortex is no longer held in check by the striatum.

Dr. Adolf Meyer, Baltimore: It is really surprising, after studying serial sections of acute encephalitis, to note how extremely difficult it is to find anything distinctive and localizing, with the hope of obtaining a picture similar to the poliomyelitis symptomatology.

I should like to challenge those who speak so glibly of thalamic and striatic lesions to show evidence of more definitely circumscribed disorders.

One of my standard serial cases is of the dystonia type, without having been able to satisfy myself with regard to the localization of the lesion apart from some diffuse atrophy of the cerebellum. Through this and some other cases of disturbance of that kind, I have reached the conclusion that before I shall accept any definite localizing interpretation of these conditions, I shall want to see accurate serial section material.

With regard to Dr. Kennedy's suggestion, it is very important not to assume that everything resembling actions is going to be psychogenic. Even if we want to explain it in psychogenic formulation, that does not mean that we are actually to handle it as if it were purely and exclusively psychogenic

because it can be formulated to some extent that way. Before we assume a focal lesion in the midbrain accounting for localized tics, we must obtain additional knowledge.

DR. GOODHART, in closing: My purpose in showing two reels on the screen presenting two groups of cases, was to demonstrate more clearly that the motor phenomena seen in encephalitis bear a striking resemblance, indeed in some cases are identical, in character and form with those observed in familial disease entities.

The revelations of such studies force on the conception that the comparatively minute pathologic changes in structures that studies of the brain in these cases reveal, do not give us the entire picture of the underlying pathology. On the other hand, the varied physical manifestations that we classify as clinical entities must depend on structural changes of a far more diversified nature than the simple and oft-times minutely localized histopathologic findings. For example, I may refer to the case of the young woman in the series demonstrating postencephalitic deformities of motion; not only did we see the typical parkinsonian posture and attitude develop within less than a year in the course of postencephalitic symptoms, but we observed the changes suggesting advanced age in her general make-up, particularly striking in her facial expression. The changes were indefinable but were most instructive in their associative relationship.

It is hardly conceivable that in such cases the whole pathology lies within the basal ganglions. The thyroid and hepatic changes so often found associated with focal lesions of the central ganglions demand a more generalized study of the visceral, the sympathetic and endocrine systems in their relationship to the central nervous system.

As Dr. Kennedy says, a study of the pictures demonstrating the deformities of motion calls for a revision of our conception of the localization of the pathologic area. He refutes the idea that tics are necessarily cortical in origin, and I think our demonstration sustains his contention.

As to nomenclature, it may be recalled that Dr. Tilney and myself, in presenting a series of cases by means of bradykinetic analysis, called attention to the need of a revision in our descriptive terms for recording deformities of motion.

PYRAMIDAL AND EXTRAPYRAMIDAL SYSTEM INVOLVEMENT IN EPIDEMIC ENCEPHALITIS *

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This report is based on an analysis of 100 consecutive cases of epidemic encephalitis from the records of the New York Neurological Institute Dispensary and Hospital and the Montefiore Hospital, New York. A considerable number of these cases were personally examined by the writers. The object was to determine the frequency of the association of pyramidal and extrapyramidal system involvement in a representative group of cases from both dispensary and hospital practice.

TABLE 1.—CLASSIFICATION OF ONE HUNDRED CASES OF EPIDEMIC ENCEPHALITIS

		Number	Total
Basal Ganglion Types:			
Paralysis agitans (basal ganglion involved)		31	
Paralysis agitans with cranial nerve palsies (basal ganglion, midbrain and pontile nuclei)		8	
Paralysis agitans with pyramidal tract involvement:			
Unilateral, left	5		
right	5		
Bilateral	2		
		12	
			51
Cerebral Types:			
Hemiplegic (pyramidal)		3	
Quadriplegic (pyramidal)		1	
Posterior central gyrus type with pyramidal signs		1	
Diffuse cerebral type with bilateral pyramidal signs		2	
Psychotic (no pyramidal signs)		1	
			8
Brain Stem Types:			
Cranial nerve type		3	
Cranial nerve type with pyramidal signs		4	
Choreiform type with no pyramidal signs		2	
Myeloradicular type with pyramidal signs		1	
Miscellaneous group (interbrain and midbrain types; somnolent and mild transient cranial nerve types)		31	
			41

Of the 100 cases analyzed (Table 1), twenty were from the dispensary and eighty from the hospital service. While the dispensary cases were of a milder type, there was little difference between them, and no distinction is drawn between these two types of patients. The time of the examination varied with regard to the duration of the disease. Of the pyramidal group (Table 2), six were examined in the first month, two in the second, three in the third, two in the fifth, one in the seventh, three in the tenth, and one each in the eleventh, fifteenth, nineteenth, twenty-second, twenty-fourth and forty-second months. It will be seen that thirteen of the twenty-four patients

* From the Neurological Institute and Neurological Service of Montefiore Hospital, New York.

TABLE 2.—THE PYRAMIDAL SIGNS IN TWENTY-FOUR CASES OF EPIDEMIC ENCEPHALITIS

Name	Age	Sex	Interval Between Original Acute Onset and Present Examination	Pyramidal Signs	Type of Encephalitis
I. S.	40	M	2 years; late recrudescence 3 months previous to examination	Left Hoffmann Abdominal, R. > L.	Paralysis agitans
R. E.	17	F	1¼ years	Knee reflex, R. ++, L. + Right Babinski	Cranial nerve type and choreiform movements
A. P.	11	F	1 10/12 years	Upper reflexes, R. > L. Knee reflex, R. > L. Ankle reflex, R. +, L. + Right clonus	Paralysis agitans
M. G.	42	F	1¾ years	Right hemiplegia Knee reflex, R. > L. Ankle reflex, R. > L. Right Babinski	Paralysis agitans
M. W.	49	M	7 months	Knee reflex, R. > L. Right ankle clonus Abdominal, L. > R.	Cerebral; central gyrus and parietal region; sensory aphasia
W. G.	33	M	11 months	Upper extremities, R > L. Abdominal dim., L. > R. Knee reflex, R. > L. Ankle reflex, R. > L. Bilateral ankle clonus Bilateral Babinski	Cerebral type
A. F.	28	F	3 months	Knee reflex, R. > L. Ankle reflex, R. > L. Right Hoffmann Abdominal, L. > R.	Paralysis agitans
M. S.	30	F	1 month	Knee reflex, L. > R. Ankle reflex, L. > R. Bilateral Babinski and Bilateral ankle clonus Abdominal and Epigastric absent	Paralysis agitans
F. D.	20	F	1 month	Knee reflex, R. > L. Ankle reflex, R. > L. Babinski, R. +, L. 0 Right abdominal absent	Hemiplegic
E. T.	30	F	5 weeks	Double Hoffmann Knee reflex, L. > R. Ankle reflex, L. > R. Babinski on left, on right ? Abdominal, R. > L.	Myeloradicular type and myoclonia
A. G.	21	F	3 months	Knee reflex, L. > R. Ankle reflex, L. > R. Abdominal, R. > L.	Cranial nerve and somnolent type
E. J.	6½	F	5 months	Right Babinski Abdominal, L. > R.	Paralysis agitans
A. E.	30	M	2 months	Knee reflex, R. > L. Ankle reflex, R. > L. Right Babinski Right abdominal absent	Hemiplegic
T. S.	37	F	1 month	Ankle reflex, L. > R. Knee reflex, L. > R. Left Babinski Abdominal, R. > L.	Paralysis agitans

TABLE 2.—The Pyramidal Signs in Twenty-Four Cases of Epidemic Encephalitis—(*Continued*)

Name	Age	Sex	Interval Between Original Acute Onset and Present Examination	Pyramidal Signs	Type of Encephalitis
M. D.	34	F	10 months	Bilateral pyramidal signs Bilateral Babinski Knee reflex, L. > R. Left Hoffmann	Bilateral optic neuritis cerebral type
I. G.	11-12	M	14 weeks	Knee reflex, L. > R. Left Babinski Left ankle clonus	Paralysis agitans type
I. S.	45	M	10 months	Knee reflex, L. > R. Ankle reflex, L. > R. Babinski, left +, right 0 Left ankle clonus	Asthenia and hemiplegic type
L. R.	19	F	.....	Babinski, left +, right 0 Oppenheim, left +, right 0 Knee reflex, L. > R. Ankle reflex, L. > R.	Cranial nerve type
A. S.	11	M	10 months	Knee reflex, L. > R. Ankle reflex, L. > R. Left Babinski Abdominal, R. > L.	Paralysis agitans
O. A.	37	F	5 months	Knee reflex, L. > R. Ankle reflex, L. > R. Babinski, left +, right 0 Abdominal, both gone	Midbrain, cranial nerve type
B. W.	38	M	3 weeks	Knee reflex, R. = L. Abdominal, R. > L. Babinski, left +, right 0	Paralysis agitans and cranial nerve type
S. B.	29	M	2 months	Upper extremities, L. > R. Ankle reflex, L. > R.	Paralysis agitans and slight choreiform
J. B.	..	..	3 weeks	Right Babinski Left ankle clonus Ankle reflex and knee reflex markedly exaggerated	Somnolent, bilateral, pyramidal and cranial nerve
L. R.	25	F	3½ years	Knee reflex and ankle reflex markedly exaggerated Right Babinski Abdominal, L. > R.	Paralysis agitans

(54 per cent.) with pyramidal cases were examined within five months, and six (25 per cent.) in the first month of the disease. These figures seem to indicate that some of the early pyramidal signs disappear as the acute stage passes off.

Not infrequently instances were found in which a recrudescence occurred months or over a year after the original attack. It seems that we are dealing in such cases with an acute lighting-up of a dormant process; or the so-called recrudescence is the result of a functional derangement caused by a gradually developing chronic inflammatory glial reaction.

We have included in the list of cases of the pyramidal tract group only those which showed a number of unequivocal signs of a lesion of the corticospinal pathway (Table 2). Merely the exaggeration of one

knee reflex over its fellow, we did not deem sufficient; but where the corresponding ankle reflex was also increased and the homolateral abdominal reflexes were diminished, we felt justified in assuming the existence of a pyramidal lesion. The great majority of patients also exhibited the extensor reflex of the big toe (Babinski) and its confirmatory reflexes. No use has been made of pathologic associated movements in the detection of pyramidal tract disturbance, because the frequently coexistent striatal lesions have rendered this group of symptoms valueless.

It will be noted that pyramidal tract involvement occurred in twenty-four cases (24 per cent.). In twelve of these (50 per cent.) extrapyramidal involvement coexisted. On the other hand, in the fifty-one extrapyramidal or striatal instances (omitting the choreiform types) twelve (23 per cent.) showed coexisting pyramidal disturbance (Table 1).

J. Ramsay Hunt [1] speaks of this association as the palliopallidal form of paralysis, and in a series of twenty-five instances of striatal encephalitis he noted it in three, that is, 12 per cent., as contrasted with our 23 per cent.

We believe this unusual combination should be stressed. We wish to point out, however, that the pyramidal and extrapyramidal signs do not always appear at the same time. In some of the patients, either after an apparent cure lasting from several months to a year or after an equally long stationary period, signs of basal ganglion involvement made their appearance. In other instances the pyramidal tract signs persisted, and after an equally variable period the extrapyramidal signs appeared. One should, therefore, suspect encephalitis in a young person with a paralysis agitants syndrome which has developed rapidly. If, in addition, there are signs of *pyramidal pathway disturbance,* one is almost certain that the combined picture is one of epidemic encephalitis. As a history of encephalitis is not infrequently difficult to elicit, this is even more important.

Grossman [2] has well said that "barring an occasional case of disseminated sclerosis or of diffuse cerebral arterial sclerosis, encephalitis seems to be the one disease that most frequently shows lesions involving the basal ganglions and the pyramidal tracts," which is explained by the multiplicity of lesions occurring in this disease.

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1. Acute Epidemic Encephalitis—An Investigation by the Association for Research in Nervous and Mental Diseases, New York, Paul Hoeber, 1921.

2. Grossman, Morris: Sequels of Acute Epidemic Encephalitis, J. A. M. A. **78**:959 (April 1) 1922.

THE STRIOCEREBELLAR TREMOR

A STUDY OF THE NATURE AND LOCALIZATION OF THE COMBINED FORM OF ORGANIC TREMOR *

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NEW YORK

All forms of tremor movement bear a certain fundamental resemblance to one another, although finer differences are distinguishable. We recognize, for example, tremors which are fine or coarse, rapid or slow, rhythmical or arrhythmical. From the etiologic standpoint many varieties are described by systematic writers, and it is surprising how few disorders of the nervous system run their course without the accompaniment of tremor. Indeed, a physiologic form is recognized, and a slight tremulousness is a common result of muscular fatigue.

In spite of recent advances in neuropathology and a better understanding of the correlation of structure and function in the central nervous system, the anatomic basis of tremor is by no means settled at the present time.

Many organic affections of the nervous system are associated with tremor, more especially those involving the cerebellum, the corpus striatum and their connections with the brain stem. Among these, paralysis agitans, multiple sclerosis, pseudosclerosis and progressive lenticular degeneration are especially deserving of mention because of the constancy and characteristic nature of the tremor and the localization of the lesions.

In the organic group of tremors two distinct clinical types may be differentiated; one, the tremor of repose; the other, appearing only during the course of movement itself, the so-called "intention tremor." In the present study I shall consider more especially the relation of these two forms of tremor to the corpus striatum and the cerebellum and their occurrence as a combined form—the striocerebellar tremor.

CHRONIC PROGRESSIVE CEREBELLAR TREMOR (DYSSYNERGIA CEREBELLARIS PROGRESSIVA)

Some years ago, under the title [1] given above, I directed attention to a chronic progressive tremor disturbance, which I regarded as a definite clinical type of organic nervous disease.

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* Read in abstract at the Forty-Eighth Annual Meeting of the American Neurological Association, Washington, D. C., May, 1922.

1. Hunt, Ramsay: Dyssynergia Cerebellaris Progressiva, a Chronic Progressive Form of Cerebella Tremor, Brain **37**:247, 1914-1915.

The symptomatology was characterized by generalized intention tremors, which began as a local manifestation and gradually extended to the whole voluntary muscular system. Associated with the tremor disturbance were the characteristic manifestations of cerebellar disease: dyssynergia, dysmetria, adiadokokinesis, hypotonia and asthenia. In all other respects the neurologic examination was essentially negative.

Three cases were described in my original communication, all similar in their general course and symptomatology, differing only in degree and the duration of the disease.

The intention tremor, which was the striking and characteristic symptom of the disease, began in one extremity and progressed slowly, involving gradually and successively the remaining portions of the body. There was a coarse, irregular atactiform shaking on attempting any movement. The tremor movement was slow, ranging from three to five vibrations a second. Both the rate and amplitude were increased by mental and physical activity. It was diminished or ceased entirely in a relaxed or recumbent posture and was consequently absent during sleep. After paroxysms of prolonged motor agitation some after-tremor persisted, even during rest.

The whole course of the disease was chronic and slowly progressive, and the motor life became more and more restricted. Once established, the tremor never disappeared except during rest.

The clinical picture in this group of cases was characteristic of a disturbance of the cerebellar mechanism. Furthermore, the disorder progressed gradually, in the manner of an organic degenerative disease, and on the basis of these clinical observations I postulated a progressive degeneration of certain cells or fiber systems of the cerebellar mechanism as the probable pathology of the disease, the exact localization of which must await the results of pathologic investigation.

Since this statement was made my conception of the nature and symptomatology of this group of cases has been considerably enlarged by further clinical and pathologic investigations.

I have had occasion to observe another clinical group which combined the symptomatology of dyssynergia cerebellaris progressiva (progressive cerebellar tremor) and myoclonus epilepsy.[2] There was the progressive dyssynergia and intention tremor characteristic of a cerebellar disorder, in association with epilepsy and myoclonus. In this group, as in the original group of cases uncomplicated by myoclonus epilepsy, the extremities showed the greatest degree of involvement.

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2. Hunt, Ramsay: Dyssynergia Cerebellaris Myoclonica — Primary Atrophy of the Dentate System; a Contribution to the Pathology and Symptomatology of the Cerebellum, Brain **44**:490, 1921.

The association of cerebellar dyssynergia and myoclonus epilepsy suggests the occurrence of two independent nervous disorders in one person. Such a combination, however, is rare and shows merely a predisposition to the two disorders in the same person; it does not necessarily indicate any essential relationship between them.

On the other hand, little is known at the present time of the pathology and localization of myoclonus. Its occurrence, therefore, in conjunction with a special type of cerebellar disease, of which I have observed six cases, is not without interest, and may have a deeper pathologic significance than might first appear.

This form of dyssynergia, which I termed dyssynergia cerebellaris myoclonica, was also observed in association with Friedreich's ataxia, a combined cerebellospinal involvement, which is not infrequent in the history of cerebellar system disease. In one of these cases studies of the pathology were made, which throw considerable light on the origin and anatomic basis of the cerebellar portion of the symptomatology. As a result of these investigations, the progressive dyssynergia and intention tremor was referred to an atrophy of the efferent dentate system of the cerebellum, and this system was regarded as the essential neural mechanism underlying the production of the cerebellar or intention tremor.

Pathologic Changes.—The important and essential lesion of the cerebellum was an atrophy of the motor cells of the corpus dentatum and the superior cerebellar peduncles. There was no atrophy of other cerebellar systems, and none of the nucleus ruber. The lesions of the cerebellar mechanism were, therefore, confined to the short and important internuncial common pathway which conveys efferent impulses from the cerebellum to the spinal cord.

The relation of organic tremor to the cerebellar mechanism was emphasized some years ago by Gordon Holmes.[3] He reported a series of cases of organic cerebral disease which, while in many respects dissimilar, had one symptom in common—a tremor, which presented the following characteristics:

> In every case it was found that the patient was unable to voluntarily check the movement for more than the shortest space of time, and often the attempt to inhibit it only resulted in increase or accentuation of the range. In addition to the tremor proper, which may be described as static, as it occurred independently of voluntary or reflex movement of the part, volitional movement of the limb affected was in every case complicated by an irregularity of the intention tremor type, as met with in disseminated sclerosis, that is, the range of deviation from the direct line of the movement increased as the completion of the act purposed was approached.

3. Holmes, Gordon: Certain Tremors in Organic Cerebral Lesions, Brain **27**:327, 1904.

In no case did the tremor persist during sleep. It also ceased when the limb involved lay at complete rest, so that each of its segments was individually supported. The lesions, so far as could be determined, were localized in the dorsal or tegmental region of the midbrain, sometimes extending forward into the optic thalamus. Holmes described in detail the nucleus ruber and its connections and concluded with the statement that "It seems from anatomical and other considerations that it is to some involvement of this system that the tremor above described is to be attributed."

The general resemblance of the type of tremor described by Holmes to that presented by my patients with dyssynergia cerebellaris progressiva (chronic progressive cerebellar tremor) is evident. In the group of cases which I described, however, the tremor was monosymptomatic, while in the group described by Holmes it was one among other organic symptoms.

The coexistence in this group of cases of symptomatic features characteristic of both striatal and cerebellar tremor may be pointed out, and will be discussed more in detail. The following case was originally described in my clinical study of dyssynergia cerebellaris progressiva (chronic progressive cerebellar tremor). Death occurred thirteen years after the onset of the tremor, and opportunity was afforded for histologic study of the central nervous system.

REPORT OF A CASE

Chronic progressive striocerebellar tremor associated with cirrhosis of the liver (tremor type of the hepatocerebral degeneration).

History.—A woman, aged 40, had volitional tremor of the left arm. One year later a similar tremor of the right arm developed, which was followed by gradual extension to the musculature of the head, trunk and lower extremities. The clinical picture was one of generalized coarse, ataxic tremor when the patient attempted to move, which ceased during rest. A study of the motility revealed also a disturbance of the cerebellar function. There was dysmetria, dyssynergia, hypotonia, adiadokokinesis and asthenia; otherwise neurologic examination was negative. The chief symptom was generalized dyssynergia with tremor movements on intention.

Tremor: When the patient lay in the recumbent posture with the body completely relaxed and the head supported by a soft pillow, there was no movement.

The slightest attempt at innervation, such as fixation of the eyes, a movement of the hand, a simple flexion of the extremities, or even attempts to speak or smile, produced tremor, which was increased by any attempt at repression.

Mental excitement and effort also aggravated the tremor. The automatic act of respiration alone, when the patient was quiet and relaxed, did not produce a tremor; however, during the more violent exacerbations of tremor the respiratory movements were sometimes jerky and arrhythmical.

The favorite position of the patient while sitting was leaning forward, resting the head, arms and upper portion of the body on a table. In this

position she often remained for long periods perfectly quiet and relaxed, unless she was questioned or her attention attracted, when more or less violent tremors immediately resulted. While sitting quietly in a chair before the examiner with the arms resting on the lap, the tremor was confined to nodding and shaking of the head, some facial movement and oscillations of the body, but mental excitement or a slight attempt at voluntary innervation, such as speech or movements of the fingers, apparently disturbed the balance and adjustment of the patient, and violent tremors resulted.

There was no true nystagmus. If, however, the tremor was checked by holding the head, tremor occasionally reappeared in the eyes. Such oscillations, however, were not obtained by fixation of an object with the eyes. The overflow of tremor also occurred when movements of an upper or lower extremity were forcibly checked.

The tremor of the extremities was of the volitional or intention type, and consisted of a coarse ataxic shaking and tossing of the extremities rather than a true rhythmical tremor, although in certain positions this ataxic shaking assumed a more or less rhythmical character.

On attempting to place the index finger on the tip of the nose, the arm was jerked and thrown about with ataxic violence, the motor agitation subsiding and becoming less severe when the object of the movement was finally attained. On attempting to place the heel on the knee in the recumbent posture, the same coarse volitional disturbances appeared, and when the leg was elevated, as in the arm, a violent ataxic tremor developed. Closure of the eyes had no appreciable influence on the extent or character of the movements.

Speech was slow and scanning and was frequently broken and interrupted by violent explosive efforts and utterances. Under excitement these brusque explosive discharges rendered it almost unintelligible. The speech disturbance was evidently caused by the same disharmony which characterized the other muscular efforts. During the act of articulation there were associated tremor-like contractions of the facial movements, and the tremor of the head was much exaggerted.

On standing, the general tremor was much increased, the legs shook, the trunk oscillated, the head was in constant movement, and the arms were tossed and hurled about in the most bizzare fashion. Chewing and swallowing aggravated the tremor, which added to the difficulty of taking nourishment. Static equilibrium was well maintained even on a narrow base, and closure of the eyes in this position had no apparent effect on posture or the intensity and character of the tremor. For some years all finer movements of the hands had been impossible, and the handwriting had been reduced to illegible scrawls and scratches. If the patient had had an exciting or fatiguing day, some after-tremor might persist for several hours, even during the period of rest.

Hypotonia: The muscles were well developed and free from atrophy. They were, however, soft and flabby to the touch and there was a definite hypotonia. The joints were relaxed and flaccid and were sometimes overextended. The Stewart-Holmes sign of hypotonia was also constantly present in the upper extremities, that is, the failure of rebound when flexion of the arm was resisted and suddenly relaxed.

Dysmetria and Dyssynergia: There was a distinct disturbance of ability to measure, regulate and harmonize voluntary movements in the extremities.

Adiadokokinesis: This was present in both upper extremities.

Sensation: The general sensations both superficial (touch, pain and temperature) and deep (muscular and articular) were normal. There was no demonstrable defect in the ability to distinguish the relative difference of weights placed in the hands.

Vision, the sense of smell, taste and hearing were normal.

Reflexes: The tendon reflexes of the upper extremities (supinator, biceps and triceps) were present, not exaggerated, and equal on the two sides. The reflex of the jaw was present and not exaggerated. Knee and ankle reflexes were present on both sides; they were of equal intensity and not exaggerated. The abdominal reflexes were present and equal. The plantar reflex gave a normal flexor response on both sides, and the Babinski reflex had not been demonstrable during the many years of observation in the hospital.

Cranial Nerves: The pupils were equal and reacted promptly to light and accommodation; the pupillary skin reflexes were normal.

Ophthalmoscopic examination revealed normal optic nerves; no signs of neuritis or pallor of the disk. The ocular excursions were normal; there was no true nystagmus. The innervation of the facial muscles, the muscles of mastication, soft palate and tongue were normal, but produced marked tremor disturbances.

Course of Illness.—The patient remained under observation in the Montefiore Home up to the time of her death, Nov. 1, 1920. During this period there were no essential changes in the character of the clinical picture as originally described in my paper in 1914. The tremor disturbance, which was the dominating symptom of the disorder, persisted and slowly increased in severity. There were no paralysis, no sensory disturbances, no spasticities or rigidities. At no time did I observe any paralysis of automatic associated movements of the paralysis agitans type—which was in harmony with the absence of all muscular rigidity. The tendon and skin reflexes were normal and equal on the two sides. The Babinski reflex was absent. There were no areas of pigmentation of the skin or of the corneal margin. With the progress of the disease mental deterioration and emotional instability were noted. The mental changes, however, were not of a severe character; memory and judgment were retained, and there were no delusions or hallucinations. In the emotional sphere there was at times depression, and occasionally a slight degree of euphoria was noted.

Necropsy Findings.—Necropsy revealed the typical lesions of pseudosclerosis associated with nodular cirrhosis of the liver, similar in appearance to that described by Wilson[4] in progressive lenticular degeneration.

The central lesions were most marked in the lenticular nuclei, the cerebellum and pons varolii. In all of these neural structures there was some breaking down with cavity formation. There was, however, no evidence of inflammatory reaction. The Alzheimer glia cells which characterize the histologic lesions of pseudosclerosis were distributed extensively throughout the brain. There was no degeneration of the spinal cord.

COMMENT

The character of the pathologic lesions in this case—the nodular cirrhosis of the liver and the histologic changes in the central nervous system—serve to identify it with the group of the hepatocerebral

4. Wilson, Kinnier: Progressive Lenticular Degeneration: a Familial Nervous Disease Associated with Cirrhosis of the Liver, Brain **34**:290, 1912.

degeneraions.[5] The tremor character of the clinical picture, however, distinguishes this from the other recognized clinical types, namely, the progressive lenticular degeneration of Kinnier Wilson, the tetanoid chorea of Gowers, the pseudosclerosis of Westphal and the dystonia lenticularis as described by Thomalla [6] and Wimmer.[7] I would, therefore, regard it as a tremor type of the hepatocerebral degenerations.[8] I have considered this phase of the subject elsewhere, and I refer to it because of the peculiar nature of the tremor, which in the light of the clinical features and pathologic findings may be interpreted as a combined striocerebellar tremor. There were well marked lesions in the lenticular neuclei on both sides and extensive involvement of the cerebellum and midbrain as well. The lesions, therefore, were not only distributed in the corpus striatum and the cerebellum, but also in the region of their connections with important nuclei of the brain stem. The lesions of pseudosclerosis, because of their diffuse character, are not adapted to any exact correlation of structure and function. There can be no question, however, as to the predominating involvement of the striatal and cerebellar mechanisms in this case and to the dominating importance of tremor (striocerebellar) in the clinical picture.

Therefore, the small clinical group which I isolated some years ago as chronic progressive cerebellar tremor (dyssynergia cerebellaris progressiva) may be modified as a result of subsequent pathologic study. In one group, dyssynergia cerebellaris myoclonica, the cerebellar tremor is part of a general cerebellar disorder and may be correlated with an atrophy of the efferent dentate system of the cerebellum. In the other group the tremor disturbance is not purely cerebellar, but is a mixed striocerebellar tremor associated with the central lesions of pseudosclerosis (tremor type of the hepatocerebral degeneration). It is probable that further pathologic investigations will shed still more light on this interesting and comparatively rare group of organic nervous disorders.

THE RELATION OF THE STRIATAL MECHANISM TO TREMOR (STRIATAL TREMOR)

The relation of tremor to the striatal mechanism is now an accepted fact of symptomatology. There may be differences of opinion as to the

5. Hall, H. C.: La degenerescence hepato-lenticulaire, maladie de Wilson-pseudo-sclerose, 1921, p. 160.

6. Thomalla: Ein Fall von Torsionsspasmus mit Sektions befund und seine. Beziehung zur Athetose Double, Wilson's Krankheit und Pseudo-Sclerose, Ztschr. f. d. ges. Neurol. u. Psychiat. **41**:311, 1918.

7. Wimmer: Etudes sur les syndromes extra-pyramidaux, spasme de torsion progressif infantile, Rev. neurol. **28**:952, 1921.

8. Hunt, Ramsay: The Tremor Type of the Hepato-Cerebral Degeneration, Trans Assn. Am. Phys. **37**: 1922.

exact character of the tremor and the rôle of this organ in its production, but practically all authorities agree that a rhythmical tremor is one of the cardinal symptoms of the corpus striatum.

Kinnier Wilson was among the first to establish this relationship in his analysis of the symptomatology of progressive lenticular degeneration.[4] Previous to this, however, a number of isolated reports had shown that rhythmical tremor of the paralysis agitans type occasionally follows striatal lesions (Demange,[9] Rhein and Potts[10]).

According to Wilson, the tremor of progressive lenticular degeneration is slow, consisting of from four to six oscillations to the second, and is increased both by voluntary movement and psychic stimuli. It ceases during rest and is more marked in the distal portion of the extremities. The same character of tremor was noted by Sawyer, Cassirer[11] and Pollock[12] in cases of Wilson's disease. In Stoecker's[13] case, however, while the tremor was increased during movement and was therefore an action tremor, it was also present at rest (tremor of repose). This combination of a tremor of repose and action tremor will be referred to later in the discussion on the combined form of striocerebellar tremor.

In 1915, I described as primary atrophy of the pallidal system[14] a group of cases with characteristic pathologic lesions presenting the symptomatology of paralysis agitans. In one case of juvenile type, in which the disease had existed for twenty years, histologic examination was made of the central nervous system, with special reference to the corpus striatum, the cerebellum and their connections with the brain stem. As a result of these studies, I concluded that juvenile paralysis agitans is a system disease, the essential lesion of which is atrophy of the large motor cells of the corpus striatum (pallidal system).

In this case the lesion was strictly limited to the essential motor system of the corpus striatum. The tremor, therefore, may be regarded

9. Demange: Contribution a l'études tremblements prae et post-hemiplegique, Rev. de méd. **2**:371, 1883.

10. Rhein and Potts: Post-Apoplectic Tremor; Symmetrical Areas of Softening in Both Lenticular Nuclei and External Capsules, J. Nerv. & Ment. Dis. **46**:757, 1917.

11. Cassirer: Ein Fall von Progressive Linsenskern Erkrankung, Neurol. Centralbl. **32**:1284, 1913.

12. Pollock: The Pathology of the Nervous System in a Case of Progressive Lenticular Degeneration, J. Nerv. & Ment. Dis. **46**:401, 1917.

13. Stoecker: Ein Fall von Fortschreitender Lenticular Degeneration, Ztschr. f. d. ges. Neurol. u. Psychiat. **15**:251, 1913.

14. Hunt, Ramsay: Primary Atrophy of the Pallidal System. A Contribution to the Functions of the Corpus Striatum, Brain **40**:58, 1917; Arch. Int. Med. **22**:647, 1918.

as representative of the striatal type of tremor. It was slow and rhythmical with from four to six vibrations a second and was increased by mental excitement and physical activity. It was present during rest and was therefore a tremor of repose. It was increased at the beginning of purposive movements, but was not of the intention tremor type, and ceased entirely toward the end of an intentional movement; and while the tremor was increased during movement, there was not the atactiform uncertainty and other cerebellar components of the true intention tremor.

The tremor disturbance was generally distributed, and all of the extremities were affected, including the head. Typical pill-rolling movements of the fingers were present; if suppressed in one part, the tremor made its appearance in some other part of the body.

In the earlier years of the disease, the tremor was coarse and at times violent, more especially during the stress of emotion or attempted movement. As the malady progressed and the extremities became fixed by postural rigidities, the severity of the tremor gradually lessened.

The clinical characteristics of tremor resulting from a lesion of the corpus striatum may therefore be summarized as follows: Tremor occurs spontaneously and is slow and rhythmical in character. It may be increased by mental excitement and physical activity, and persists during rest (tremor of repose). If checked in one extremity, there is a tendency for its reappearance in other parts of the body (overflow of the tremor). Although subject to variation, it has the same tempo in all parts where tremor is present.

The mechanism underlying the production of striatal tremor is still one of the mooted questions of pathologic physiology. I regard it as a release phenomenon resulting from loss of striatal control and the expression of spontaneous activity in certain infrastriatal centers of the extrapyramidal system. It is, therefore, primarily a disorder of motility in the paleokinetic sphere and in this sense analogous to chorea and athetoid movement.

In a previous study of the corpus striatum,[15] evidence was presented showing that the corpus striatum is a motor organ for the control of automatic associated movement, just as the rolandic area controls movement of the isolated synergic type. Two neural systems were recognized in the striatum: a large cell motor system (pallidal system) controlling movements of the automatic associated type, and a small cell system (neostriatal system) exercising an inhibitory and coordinating function. Destruction of the pallidal system was identified with the

15. Hunt, Ramsay: The Efferent Pallidal System of the Corpus Striatum. A Consideration of Its Functions and Symptomatology, Trans. Am. Neurol. Assn., 1917, p. 10; J. Nerv. & Ment. Dis. **46**:44, 1917.

syndrome of paralysis agitans and loss of the neostriatal system with chorea. The variegated symptomatology of this region was therefore attributed to an admixture of the clinical manifestations of these two syndromes.

The corpus striatum through the medium of its efferent pallidal system has important connections with subsidiary motor centers of the midbrain and hypothalamic region, among the more important of which may be mentioned the nucleus ruber, the locus niger and the corpus subthalamicum. Any break in the striospinal system causes a paralysis of automatic associated movement and releases the infrastriatal motor centers from control, with the development of rhythmical tremor and hypertonicity (rigidity). The mechanism is similar to that of the development of clonus and hypertonicity (spasticity) following lesions of the rolandic area of the cerebral cortex (neokinetic system).

Tremor and clonus resemble one another in many respects, and both tremor manifestations are, I believe, referable to the kinetic system, one due to a release of the paleokinetic, and the other of the neokinetic, mechanism.

Relation of Striatal Tremor to the Kinetic System.—I have already presented at some length my views on the duality of function of the motor system.[16] According to this conception, the efferent nervous system, both cerebrospinal and vegetative, consists of two components which are anatomically and physiologically distinct. One component is the kinetic system, which controls the movement proper; the other is the static system, which regulates the postural function of the contractile mechanism.

According to this conception, the kinetic system controls the disk mechanism of skeletal muscles, and the static system the sarcoplasm. The essential function of the kinetic system and the fibrillar structure of the muscle fiber is movement. The essential function of the static system and the sarcoplasmic structure of muscle is fixation or posture.

In the symptomatology of motor disorders certain manifestations are referable to the kinetic system and others to the static system; and while in any disorder of motility both systems participate, it is usually possible to indicate one or the other as the essential factor involved. These two systems of motility also play a rôle, I believe, in the production of organic tremor. The striatal tremor is related to the kinetic system and the cerebellar tremor to the static mechanism.

Relation of the Cerebellar Mechanism to Tremor (Cerebellar Tremor).—For many years, tremor has been a well recognized, although

16. Hunt, Ramsay: The Static and Kinetic Systems of Motility, Arch. Neurol. & Psychiat. **4**:353 (Oct.) 1920. The Static or Posture System and Its Relation to Postural Hypertonic States of the Skeletal Muscles, Neurol Bull. **3**:207, 1921.

inconstant, symptom of cerebellar disease. Cerebellar tremor is of the so-called intention type, resembling in character the tremor of multiple sclerosis. It is associated with other manifestations of a disorder of cerebellar function, for example, dysmetria and adiadokokinesis, and is only an extreme manifestation in the extremities of the underlying disorder of muscle synergy (asynergia) which is the fundamental symptom of a cerebellar disease. It has the character of a coarse ataxia and in contradistinction to the tremor of paralysis agitans is always absent during rest. It is, therefore, never a tremor of repose. The tremor manifestation is coarse and arrhythmical, beginning with movement and increasing as the object is approached. The latter feature of the cerebellar tremor should be particularly emphasized as an important difference between the so-called action tremor, which is sometimes observed during the movement of the extremity in paralysis agitans, and which diminishes and usually ceases as the object of the movement is approached. This peculiar feature of the cerebellar tremor is, I believe, dependent on the nature of cerebellar function and its relation to the static system.

According to the view which I have previously expressed, the essential central organ of the static component of the efferent system is the cerebellum. All higher impulses subserving the static or posture function of motility pass to this organ for final integration. In this conception the cerebellum is regarded as the great central ganglion for the coordination of posture synergy in contradistinction to motion synergy, which is under the control of the kinetic system.

A lesion of the cerebellar mechanism therefore disturbs the posture function of motility, the symptoms of which are dyssynergia, dysmetria, adiodokokinesis and tremor, and in this sense the cerebellar tremor is primarily postural in its origin. In any series of movement there is an accompanying series of posture which serves to reinforce the stability and accuracy of movement itself. Posture follows movement like a shadow, and without it movement loses its perfection of control and direction. The intention tremor is a particularly exquisite example of a loss of postural control. It is present only during movement and is especially active at the end of movement, when the posture system is more especially called into action. For this reason there is an atactiform tremor disturbance when an attempt is made to place the finger tip on the nose, which increases as the final act of posture is reached.

I believe, therefore, that the so-called cerebellar tremor is related primarily to a disorder of the static system, the coarse tremor movements being simply compensatory manifestations in the kinetic sphere.

Striocerebellar Tremor.—The disorder of motility to which I now direct attention presents the characteristics of both a striatal and cerebellar tremor. There is the spontaneous rhythmical tremor char-

acteristic of a striatal disorder in association with the intention tremor of cerebellar disease. Sometimes one component, sometimes the other, dominates the clinical picture, and there is also considerable variation in the degree and localization of the tremor disturbance.

In the group of cases described by Gordon Holmes, already alluded to, in which the lesions were localized in the tegmental region of the midbrain, both of these components of organic tremor were evidently present. In Case 1, in which symptoms were indicative of a vascular lesion of the midbrain, the description of the tremor was as follows:

> Except when the limb was at perfect rest and so supported that each segment bore its own weight, there was constantly slow clonic tremor of one or other part of it, but more marked at the distal than at the proximal joints. Frequently the adducted fingers were flexed and extended at their basal joints, the index finger rubbing against the opposed thumb, which was in synchronous tremor. Often it was flexion or extension of the hand at the wrist, or flexion and extension, but more frequently pronation and supination at the elbow. Movements were less frequently visible at the shoulder.
>
> Similar tremor was occasionally to be observed in the left lower extremity, and, as in the arm, more frequently at the distal than at the proximal joints. It most often took the form of extension and flexion of the foot.
>
> The character of the tremor was constant, no matter in which portion of either limb it was observed. It was remarkably regular in rate, and varied very little in amplitude, so that the movement of the limb, as long as only one group of muscles was involved, was essentially regular and rhythmical, but as more than one group was frequently at one time in action, the resultant movement was often a compound tremor. It was slow in rate, from two and a half to three oscillations per second, and of considerable amplitude.
>
> It always ceased during sleep and when the limb was allowed to lie at rest, so supported that each portion bore its own weight. If any part of the limb was allowed to hang passively (as the hand when the limb was held up by the forearm), the tremor immediately began at the most proximal joint of the unsupported portion. It was increased by any excitement or agitation on the part of the patient, and by movement of the opposite limb. The movements could only be inhibited for a very short time. Voluntary movements of the limb were complicated by wide irregularities of the intention tremor type, but there was no other affection of co-ordination.

This description is characteristic of a striatal tremor, the last sentence indicating, however, the admixture of a cerebellar element.

In the second case reported by Holmes, in which the clinical diagnosis was vascular lesion of the left side of the midbrain, the description of the tremor was as follows:

> Both right limbs were affected by tremor when they did not lie at perfect rest with their muscles completely relaxed, but it also came on when each portion of the limb was not so supported as to bear its own weight. Thus, it was rarely to be observed as the limbs lay in bed and the patient was quiet and unexcited. It might occur at any joint, but in both arm and leg was more frequent at the distal.

The most constant movement was flexion and extension of the hand or of the fingers at the metacarpophalangeal joints, but there was also often adduction and abduction of the fingers, and occasionally the thumb might be moved against and simultaneously with the forefinger. In the leg flexion and extension of the ankle was most frequently observed, also similar movement of the toes, but various movements at the more proximal joints were often present.

The tremor was generally compound, that is, it was rarely limited to one group of muscles and their antagonists, so that the limb was, as a rule, simultaneously moved at two or more joints. It never persisted long in any group of muscles. The clonic contractions of each group of muscles involved were essentially regular in rate but relatively slow, from 3 to 3½ per second, and the range of the movements was more or less regular but considerable, i. e., the tremor was slow and coarse. It ceased everywhere during sleep and was increased in amplitude but not in rate by excitement or agitation and by forcible movement of the fellow limb. The patient was unable to check it voluntarily except by allowing the limb to fall relaxed in perfect rest. There was marked intention tremor of both right limbs, i. e., abrupt deviations from the direct line of movement increasing toward the completion of the voluntary act.

Here again there is present an admixture of both the striatal and cerebellar elements of tremor.

In Case 3 the diagnosis was a left-sided cerebellar tumor with evidences of extension to the midbrain. The tremor manifestations were as follows:

From this time till his death, about three weeks later, there was almost constantly tremor in all four limbs. It only ceased during sleep, or when the muscles were quite relaxed and the limbs so supported that each segment bore its own weight. The tremor commenced at once when any of the muscles were put in tension or brought into action, as when part of either limb was allowed to hang over the edge of the bed, or when it was held up in the observer's hand. It was easily made out that the series of oscillations were due to alternate contraction of the one group of muscles and its antagonists, i. e., that the movements comprised a true tremor. If the limbs were left in a suitable position it seemed as if the tremor would persist indefinitely. It was forcible and not easily checked, and showed very little tendency to overflow into other groups of muscles when passively stopped in one set. As two or more movements might simultaneously occur, the resultant movementment of the limb was often compound. The upper extremities were more affected than the lower, and the left limbs very much more than the right. In the upper limbs the movements most frequently observed were flexion and extension or pronation and supination of the elbows, flexion and extension of the wrists, and flexion and extension or abduction and adduction of the fingers, which generally remained extended at interphalangeal joints. In the lower extremities flexion and extension of the ankles and similar movements of the toes were the most frequently present, but there were also often similar oscillations of the thigh round the hip joint and of the leg at the knees.

The tremor was in every part regular in rate, about five to six oscillations per second, and the rate did not seem to vary, no matter what part of any limb was affected. It may be described as coarse, i. e., of large amplitude

and the range of oscillation often changed in a more or less rhythmical manner, i. e., the range of successive oscillations would slowly increase to a certain point and then slowly decrease. Consequently the tremor must be described as irregular in range. He was unable voluntarily to inhibit the tremor, and its amplitude was increased by an excitement or agitation, or by forcible movement of the opposite limbs. The finer voluntary movements, too, were complicated by ataxia and irregularity of the intention-tremor type, so that he scarcely found it possible to bring his hand to his face.

The character of the tremor in Holmes' series of nine cases is similar to the tremor disturbance in my case of pseudosclerosis with cirrhosis of the liver. They all show clearly the dual symptomatology of the striocerebellar tremor. Evidences of the combined form of organic tremor may also be found elsewhere in medical literature.

The so-called Benedikt's syndrome [17] is not without interest in this relation. This is a syndrome caused by a lesion of the tegmental region of the crus cerebri, the clinical characteristics of which are third nerve palsy with crossed hemianesthesia and hemitremor of the paralysis agitans type. Pelnar,[18] in his monograph on tremor, states that an analysis of the literature shows considerable variation in the character of the tremor disturbance in Benedikt's syndrome, and that the tremor movements are often more of the intention type.

Pelnar also states that the tremor of multiple sclerosis may occasionally present the characteristics of a tremor of repose in addition to the intention element, and that this polymorphous type of tremor is especially likely to occur in pseudosclerosis. Strümpell,[19] in 1897, analyzed the symptomatology of pesudosclerosis and gave the diagnostic criteria by which it could be differentiated from multiple sclerosis. He stated that the tremor is both rhythmical and of the intention type.

Hoeslin and Alzheimer [20] in their study on pseudosclerosis state that the tremor is of the intention type but that it also occurs frequently during repose.

One of Oppenheim's [21] patients had a tremor of repose in the right hand and a tremor of the intention type in the left. In another case

17. Benedikt: Nerven pathologie **2**:74.

18. Pelnar, Josef: Das Zittern, Ztschr. f. d. ges Neurol. u. Psychiat. **8**: 1913.

19. Strümpell: Die Westphalsche Pseudo-Sklerose und über diffuse Hirnsklerose, insbesondere bei Kindern, Deutsch. Ztschr. f. Nervenheilk. **2**:115, 1897-1898.

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the tremor was described as of large amplitude, existing during repose and appearing also on intentional movement.

Nature and Localization of Striocerebellar Tremor.—From the foregoing description and quotations, I think there can be little doubt as to the existence of a tremiform disorder of motility which combines the elements of striatal and cerebellar tremor. The striocerebellar tremor appears in the course of pseudosclerosis, more rarely in multiple sclerosis, and is not infrequently observed after inflammatory, vascular and neoplastic lesions in the region of the midbrain. This is the meeting place of the efferent systems of the corpus striatum (pallidal system) and the cerebellum (dentate system), which converge and terminate in relation to the ganglionic masses of this region, of which the nucleus ruber is an especially important constituent.

Holmes was inclined to attribute the tremor disturbance in his group of cases to a lesion in the cerebellorubrospinal system, and more especially to its cerebellorubral portion. According to the view which I have expressed, this would account only for the cerebellar component. The striatal element I would refer to a lesion of the striorubrospinal system.

In the present state of our knowledge it would be unwise to limit our anatomic conceptions in the interpretation of organic tremor to the rubral connections of the corpus striatum and cerebellum. There are many other important nuclear structures in this region subserving a motor function, which Edinger has grouped under the general term, nucleus motorius tegmenti. The striatum and cerebellum both participate in the control of these subsidiary centers, so that for the present our conception should include both striatal and cerebellar components of this supraspinal mechanism. I would, therefore, modify Edinger's generalization of a nucleus motorius tegmenti to include both a kinetic and static representation—the corpus striatum controlling the kinetic component, and the cerebellum its static counterpart.

In line with this conception I would regard the striocerebellar tremor as representing a disorder of both systems, the striatal component of tremor being referable to the kinetic system and the cerebellar to the static mechanism.

CONCLUSION

In conclusion, therefore, I would postulate the existence of a combined form of organic tremor caused by the involvement of separate neural mechanisms. We recognize combined forms of palsy, central and peripheral, pallidal [15] and pyramidal, as well as combined forms of sensory disturbances due to simultaneous involvement of more than one system. In this category I would place the striocerebellar tremor.

Abstracts from Current Literature

CONCERNING MYELINIZATION IN THE CEREBRAL CORTEX.

Goichi Hirako, Schweiz. Arch. f. Neurol. u. Psychiat. **10**:275, 1922.

The work of Meynert, and particularly that of Flechsig, in which the progress of myelinization of various parts of the central nervous system was studied, was very important. Flechsig was able to distinguish about forty-five myelinogenetic centers. Myelinization of the cortex, however, has not, thus far, been very carefully studied. The material on which Hirako's study is based consisted of fifteen brains of persons ranging in age from the new-born infant to the child of 4½ years; each brain was cut in serial sections.

The intracortical myelinated structures can be divided into the tangenital fibers, Kaes-Bechterew's line, Baillarger's and Vicq d'Azyr's line, the interradial network, and the radial fibers. As tangential fibers, only those in the outermost layer, the so-called lamina zonalis of Vogt, or plexiform layer of Cajal, were included. Other horizontally running fibers, sometimes spoken of as tangential, were grouped with the deeper structures. Meynert, in 1869, regarded these fibers as having great functional importance; on the other hand, they are sometimes looked on as phylogenetically old and relatively simple structures.

Tangential fibers were found to be myelinated in the following order:

1. The hippocampus comes first with myelinization begun in the brain of a 3 weeks old child and definitely established by the age of 3 months; it is also present early in the uncus, the substantia perforata anterior, the limen insulae, and the regio olfactorio of Broca.
2. The lobulus paracentralis and upper half of the gyrus centralis anterior, at the age of 4 months.
3. Base of the gyrus frontalis superior and the lower portion of the gyrus centralis anterior.
4. Gyrus frontalis inferior and the posterior portion of the gyri orbitales.
5. The gyrus temporalis superior.
6. The gyri occipitales and cuneus.
7. The upper half of the gyrus centralis posterior and gyrus parietalis superior.
8. The gyri lingualis, temporalis inferior, posterior half of frontalis superior.
9. Gyri fusiformis, temporalis medius, and lower half of the gyrus centralis posterior.
10. In the gyri parietalis inferior, anterior half of the frontalis superior, and frontalis medius.

It will be seen that in contradistinction to the gyrus centralis anterior, the gyrus centralis posterior shows a marked delay in the formation of the tangential fibers. The areas indicated under 1, 2 and 3 belong to the primordial areas of myelinization.

Kaes-Bechterew's Line.—This can be found first at the age of 4½ years, particularly in Heschl's gyrus and its vicinity.

Baillarger's and Vicq d'Azyr's Line.—The former shows definite myelinization at 4½ years of age. At from 6 to 8 months of age, these lines are scarcely discernible. Vicq d'Azyr's line can be seen as early as the sixth month, before Baillarger's line can be found.

The Radial Fibers.—By the sixth month these scarcely approach the inner line of Baillarger, that is, the fifth or fourth cell layer of Brodmann's classification. By the eleventh month they have approached the outer line of Baillarger, or the third cell layer. The radial fibers first approach the culmen, later the sides, and finally the base of the gyrus. The U fibers myelinize at about the same time as the radial fibers.

It is not known what factors are responsible for the early topical myelinization. According to Monakow, those fibers best supplied by blood vessels show the earliest myelinization. The author thinks that this is quite in agreement with his observations. From the phylogenetic standpoint it is interesting to note that in the cat, dog, and other animals the tangential fiber development is as marked, if not more marked, than in the human brain; the author found these fibers well developed in several cases of microcephaly. It is furthermore apparent that the phylogenically older portions of the brain, such as the hippocampus and the uncus, show early myelinization. Whether functional necessity plays a big part in this is still an open question.

In conclusion the writer points out that the tangential fibers myelinize in their own way and are quite distinct from the myelinogenetic zones of other parts of the brain. Not unlike these, however, myelinization begins in isolated areas and comes to completion at about the same time of life, that is, the fourth year. The early myelinization of those areas of brain subserving smell and taste possibly results from proper nutrition and the avoidance of deleterious substances, since these functions are of particular importance to the sustenance and preservation of the individual.

WOLTMAN, Rochester, Minn.

FACIAL PARALYSIS. ALEXANDER GIBSON, Surg., Gynec. & Obst. **33**:5, 1922.

The author gives a thorough exposition of the subject of facial paralysis and calls attention to the gravity of the condition. He says that the psychic effect of the condition aggravates the physical defect. It is a lesion that disturbs the patient's whole social life, especially if it occurs in early life. Facial paralysis is divided by the author into three types: (1) supranuclear, seen so typically in the hemiplegic; (2) nuclear, and (3) infranuclear. In nuclear and infranuclear lesions the upper and lower parts of the face are, as a rule, equally involved.

The anatomy and the course and distribution of the fibers of the seventh nerve are reviewed. With regard to etiology, he considers the different types of paralysis, and in discussing treatment lays particular stress on the cases of facial neuritis or Bell's palsy. This is due to lesions below the nucleus. Active massage or galvanism to the paralyzed muscles of the face following Bell's palsy is not indicated unless they are prescribed for their psychic effect. Operative treatment is not advised for Bell's palsy, unless there is no sign of return of function after several months.

Two types of operative treatment are considered: (1) plastic, by transference of the muscles, and (2) nerve anastomosis. Strips of temporal muscle or strips of masseter muscle may be used, but do not produce as satisfactory results as nerve regeneration. In performing the plastic operations an attempt is made (1) to elevate the angle of the mouth, (2) to assist in closure of the eye, and (3) to enable the brow to be wrinkled and the eyebrow to be raised. On the average, the results may be classed at from 50 to 60 per cent. in elevation of the angle of the mouth, 30 to 40 per cent. in elevation of the lower eyelid, and 10 per cent. in action of forehead and eyebrow.

Plastic operations leave a scar which is likely to be clearly visible, and they are more ingenious than trustworthy. They are more suitable when one branch of the facial nerve has been injured or after an attempt at nerve anastomosis has definitely failed.

He reviews thoroughly the history of nerve anastomosis and gives in detail the procedure employed in a faciohypoglossal anastomosis and prefers to transfer the hypoglossal rather than the spinal accessory.

An incision is made from well up on the mastoid process down to the greater cornu of the hyoid bone. This can be readily made in one of the natural lines of flexure of the neck, and after the lapse of a few months is almost invisible. The platysma is cut through, and the interval between the sternomastoid muscle and the parotid gland is thoroughly opened up. A few strands of fibrous tissue are usually encountered and occasionally a twig from the facial nerve may be found. The nerve itself lies rather deep, a good inch (2.54 cm.) from the surface of the skin, and generally about the level of the tip of the lobule of the ear. Its direction is in general transverse. It is important, if possible, to avoid opening the capsule of the parotid, for the lobules of the gland tend to obscure the view, and there is added oozing. This stage of the operation is at times difficult. There has usually been some removal of the mastoid process, and the scar tissue continues oozing so that the field of operation is difficult to keep clean. If there has been degeneration of the nerve, its pinkish color renders it difficult to recognize, and its soft consistency makes injury easy. It also seems to stain somewhat with the oozing blood, and because of this it is not always easy to distinguish. The nerve has usually been in the field of operation for a short time before it is clearly identified. Next, it must be isolated up to the stylomastoid foramen, which may be difficult. The posterior auricular artery, which runs along the upper border of the posterior belly of the digastric muscle, seems to send off numerous fine twigs which are always ready to shed a few corpuscles in the field, and there are equally numerous venous radicles assisting in the process. Again, the nerve passes in to the deep surface of the mastoid process, and it may be necessary to remove the tip of this prominence in order to make the exposure complete, as was done by Halstead.

ADSON, Rochester, Minn.

SOME ASPECTS OF MENTAL HYGIENE. E. FARQUHAR BUZZARD, Ment. Hygiene, **6**:449 (July) 1922.

In the presidential address before the Section of Psychiatry, Royal Society of Medicine, Buzzard presented a well-balanced and logical discussion of the importance and rôle of mental hygiene in the practice of medicine. Particular emphasis is placed on the absurdity of retaining the artificial distinction between "nervous" and mental. Mental illness is a definite understandable term and is equally applicable to functional nervous disorders and to the psychoses. The difference is one of degree. Insanity should be relegated to the police court as it denotes an abnormal state which brings a person in conflict with the law. The confusion between medical and ethical principles should be cleared away. A mentally sick patient should never be regarded as a problem in ethics. At least the elementary considerations of psychopathology and psychotherapy should be taught to medical students. Buzzard feels that freudian analysis has taught some valuable lessons, but he cannot regard it as a successful system of therapeutics. Finally, the recognition of the multiplicity of etiologic factors.

the correct appraisal of fatigue, and the value of teaching constructive thinking to the patient are all stressed as essential parts of a workable doctrinnaire of mental hygiene.

Buzzard's paper is a worth-while example of sound and broad psychiatric thought. Perhaps this is all the more remarkable since his most brilliant efforts heretofore have been in the field of organic neurology. Psychiatrists should profit by studying his attitude, for in psychiatry there is always present the necessity of guarding against the development of that kind of intellectual anopsia which results from fixing the mental vision too narrowly on this or that theory to the exclusion of all other possibilities.

STRECKER, Philadelphia.

A NEW TREATMENT OF SEVERE PARAPLEGIA IN POTT'S DISEASE BY PUNCTURE OF THE ABSCESS THROUGH THE INTERVERTEBRAL FORAMEN. JACQUES CALVÉ, Presse méd. **30**:246 (March 22) 1922.

Besides the more usual orthopedic measures, two surgical methods were devised against the paraplegia in Pott's disease, which is generally caused by pressure of the tuberculous focus on the anterior surface of the cord. This writer finds that the first operative method, laminectomy, has proved unsatisfactory, affording little or no relief to the paraplegic symptoms; and that the second, "costotransversectomy" (resection of the neck of the rib and corresponding transverse process) of Ménard affects the paraplegia favorably but leaves a fistula, a serious complication.

By the new method, a hollow sound of appropriate curvature is inserted through a minimal incision close to the line of spinous processes, in such a direction that it can be made to pass through the desired intervertebral foramen and to penetrate directly into the space anterior to the anterior surface of the dura, that is, into the abscessed body of the diseased vertebra. The intervertebral foramen persisted even in severe deformities, and was sufficiently large to pass an instrument of 2 mm. diameter, avoiding the nerve and vessels. The technic is described in detail, with illustrations.

Ten such puncture operations were performed in a series of six cases. Three patients with paraplegias of long standing were not benefited. Of three patients in whom the onset had been more recent, two were symptomatically cured and one considerably improved. No ill effects were observed in any case.

HUDDLESON, New York.

ANHEDONIA. ABRAHAM MYERSON, J. Psychiat. **2**:87 (July) 1922.

Under the term "anhedonia" Myerson describes a syndrome consisting of a deficit of the normal urge to satisfy hunger, thirst, fatigue and sex inclination, and an absence or distortion of the pleasurable feelings which accompany and result from their satisfaction. There is loss of determining and motivating energy and of life objective. Excitement which may be defined as "an undifferentiated result of stimuli, whether these come from without or within," and which ordinarily has associated with it a pleasurable feeling tone, in anhedonia becomes disorganized and painful. The syndrome has been noted by Myerson after infectious diseases, particularly influenza, as a sequel of surgical operations and pregnancy, during the menopause, in males at the senium, early in dementia praecox and depressions, and sometimes as a result of complete blocking of purpose in one whose interests have been rigidly narrowed and

strongly centralized. There is perhaps some contradiction in describing anhedonia as a loss of desire and satisfaction, which gives the implication of diminished affect, and at the same time emphasizing the increased reaction to "excitement" which may have a strong emotional value as a cardinal factor in the syndrome.

STRECKER, Philadelphia.

THE DIFFERENTIAL DIAGNOSIS OF SCHIZOPHRENIA. E. FRANKHAUSER, Schweiz. med. Wchnschr. **52**:401 (April 27) 1922.

The credit for having coined the terms manic-depressive insanity and dementia praecox belongs to Kraepelin. While much opposition to this new classification was encountered, it has gradually become accepted.

The author attempts to define the term dementia praecox (schizophrenia), and to distinguish this disease from manic-depressive insanity and hysteria. To facilitate his discussion, he briefly reviews the chief symptoms of each group.

Under the heading of dementia praecox, dementia praecox simplex is the most important group. It is characterized by a disturbance of basic functions. Change in character is the fundamental factor. There is confusion of attention, lack of concentration, impairment of volition and absence of sense of duty. There may be dulness, slowness and confusion in reaction ability. Next in importance is the change in emotion, such as an inadequate reaction when sad. These affect disturbances may also be designated as a dissociation of the affect.

In manic-depressive psychosis, affect disturbances, while common, are not fundamental, and present an entirely different character. Thus in mania the entire affect life is of an even nature—the so-called manic feeling tone. It may be transferred into an elevated or jovial state, associated with self-confidence, satisfaction and assurance, but there may also be a misinterpretation of the outer world. In melancholia the affect life is of a depressive nature, with a feeling of disapproval and dissatisfaction. The entire emotional life of the dementia praecox patient is unsystematized, irregular, and seems foreign and not understood; on the other hand, the affectivity of the manic or melancholic patient is more akin to normal reactions and within our grasp.

Next in importance in dementia praecox is the so-called desultory or disconnected thinking, which terminates in a word salad or dissociation.

Among the secondary symptoms of dementia praecox are the catatonic states, practically unknown in manic-depressive insanity. Manic phases of dementia praecox and mania with schizoid tendencies occur, as do also depressions with praecox trends. It is here chiefly a matter of terms, and possibly of prognosis, as in the schizophrenias the prognosis is always of a doubtful nature.

Hallucinations and delusions are present in both manic-depressive insanity and in dementia praecox. In the latter they are more the result of dissociation, and not as in manic-depressive insanity, the expression of a disturbed affect. It is no longer held to be a fact that a certain type of delusion is characteristic of a definite psychosis. The idea of being disliked may be normal; it may be the result of a false impression. In depression, it is endogenous; in paranoias and in other delusional psychoses, it may be the result of compulsion or faulty reasoning.

The differentiation of dementia praecox from paranoia is also considered by the author. Kraepelin has narrowed the term paranoia a great deal. He has taken out the querulous type, but still considers it a form of paranoia.

In this narrowing, the paranoid type of dementia praecox has been greatly enlarged, and here also groups of affect disturbances, catatonic types, even manic reactions, have found their way. Because of the variability of this group, the paraphrenias have not been adopted in Switzerland with any enthusiasm. In the person suffering with melancholia, the cause of the ailment and mental suffering is within, while in the paranoid states the world at large is at fault.

As easy as it may be to recognize the outspoken case of dementia praecox, just so difficult is it to make a diagnosis in the milder forms. Differentiation from normal is especially difficult.

Some of the chief characteristics of the milder types of dementia praecox are: singular make-up; whimsical, shut-in, planless living; inability to use money sensibly; leaving jobs without reason, and the tendency to blame others for ill luck or misfortunes. People with dementia praecox are without any sense of responsibility, do not concern themselves with realities, will not accept facts, are intractable, stubborn, aimless and restless. They may also be depressed. Confusion of thought, attention, interest and volition is not uncommon. Apathy and indifference may be present. Kretschmer has shown that these schizoid natures are markedly inadequate, in contrast to the cyclic make-up of the manic-depressive constitution. He has gone so far as to trace these types back into the normal and determine a schizothymic or cyclothymic make-up.

The differentiation of dementia praecox from hysteria is more difficult. The diagnosis of hysteria is being made more and more rarely. All large institutions have cases of dementia praecox, epileptic equivalents, or organic disease which were formerly called hysteria. According to Aschaffenburg's definition, hysteria is characterized by the failure of stimuli to cause normal bodily reactions. Such states as "Dämmerzustande" and Ganser complex belong to the psychic forms of hysteria; and while they may be associated with organic psychosis, they indicate an hysterical mechanism. It is extremely difficult to define an hysterical character. There is inquisitiveness, lack of understanding of the rights of others, inadequacy or fantasy life. These, while characteristic of hysteria, may be schizoid or schizothymic characters. Suggestibility is always of marked importance. Egotism and untruthfulness are more closely allied to hysteria than to schizophrenia, but are not diagnostic. Pseudologia, on the other hand, is rather a symptom of schizophrenia. In dementia praecox the shut-in life is a means of satisfaction to the patient. In hysteria there is much more concern with the outside world. The emotional reactions of hysteria on the whole are understandable; they arouse the impression of being artificial and untrue. In the schizoid, though changeable, queer and disconnected, the symptoms on the whole seem to be more sincere, though entirely foreign to us. Jealousy and intrigue are as common to schizophrenia as to hysteria; prostitution is more common in schizophrenia and imbecility.

In hysteria everything is overdone, says Bumke; work as well as pleasure, love and hate, fear and anger, joy and pain, sorrow and gladness, enthusiasm and disgust; and every affect, as every mood, can suddenly be replaced by its opposite. Hysteria rests principally on an exaggerated affective basis.

The principal difference between hysteria and dementia praecox is that in hysteria the symptoms are analogous to the normal. In dementia praecox inadequate reactions chiefly associated with catatonic, paranoid or manic depressive reactions occur. A combination of hysteria and other psychoses, especially dementia praecox, presents a difficult problem. One hesitates to

make a combined diagnosis, but frequently enough such a condition presents itself. Psychopathic personalities of various types are also to be considered, especially as they may suggest a schizophrenic make-up. Patients with moral insanities comprise another important group. Here the chief characteristic is an ethical defect, a lack of conscience and duty. Many of these persons have dementia praecox with catatonic symptoms, and belong to the simple dementia praecox group. Lack of ethical qualities is not a necessary characteristic of dementia praecox, though it may be associated with it. It is more frequently seen in the manic type of manic-depressive insanity. On the contrary, it is less frequent in the depressive phases in which a high ethical character is maintained. The occurrence of chronic alcoholism in simple dementia praecox is well known. The differentiation of early general paresis from dementia praecox is not always easy, especially when no serologic data is obtained. It is of importance to note that character alteration alone may for years be the only indication of general paralysis.

In conclusion, the author states that dementia praecox is characterized by dissociation or inadequacy of the emotions. Manic-depressive insanity, on the other hand, is characterized by change of mood. Paranoia is the result of imaginative affect disturbance, and it is in this condition that misrepresentations, grandiose ideas, ideas of persecution and the like arise. The outstanding factor in all forms of dementia praecox is dissociation in the widest sense, that is, the dissociation or inadequacy of affect.

MOERSCH, Rochester, Minn.

CONCERNING DIFFUSE GLIA REACTIONS. J. L. PINES, Schweiz. Arch. Neurol. u. Psychiat. **10**:289, 1922.

There has been much speculation relative to the nature of the glial structure surrounding tumors: Does it signify a reactive process or does it mean actual tumor formation? The question essentially involves the differentiation of hyperplasia from tumor.

The writer reports a case which lends itself to the study of this problem. Complete serial sections of this brain, stained with carmine, the van Gieson, Weigert-Pal and Pal-Karmin stains, had been made under the direction of von Monakow.

A man, 50 years of age, a wine dealer, had had pneumonia at 12 years of age, and had suffered from gastric ulcer for fourteen years, from which he recovered. He was a moderate drinker. He denied venereal infection. About two years before it was noted that he suffered a change in character; while naturally good-natured and optimistic, he became depressed easily, was irritable, and recklessly speculative in business. One day while at a railroad station he suddenly lost consciousness, remaining unconscious for an hour. Following this he could speak, but remembered nothing of the attack. There was ptosis on the right, and his gait was shuffling. He improved slowly, but grew more careless in his business. He was indicted because of a false declaration, and finally was compelled to sell out. One and one-half years previously he suddenly announced that he was going to America, and without baggage departed for Geneva, only to return because he was not permitted to cross the border. One month later this was repeated, with a similar result. He became apathetic, cried a good deal, but at times was hilarious and indulged in fantasy and prevarication. One year previously, he had a second attack of unconsciousness very much like the first, preceded, however, by severe headache, dizziness and fatigue. He developed delusions of persecution; he had

some difficulty in expressing himself, and his speech became monosyllabic and dysarthric; he was disoriented, and at times had involuntary urination and defecation. Deterioration was progressive; he became indifferent to his surroundings, and was at times euphoric.

On examination, he denied ever having been depressed; he said he never drank, and that his memory was always good; he said that he made a great deal of money on speculation and that he was not sick. Calculation tests were poorly performed. He told the fable of the mule as follows: "The story is a mule which went walking, then was laden with sugar and wine. Obviously he did not drink the wine, but in a few words did that, that the water did not become cloudy, but that the whole thing was incompletely obedient. Then the men put the mule away. Ha, that was brilliant."

The pupils were moderately dilated, the right somewhat irregular and reaction to light absent; accommodation could not be tested. His tongue was tremulous and deviated to the right. There was a positive Babinski sign on the right; the left plantar reflex was doubtful. The patient was unable to stand; he had a titubating gait, placing most of his weight on the left heel; there was a tremor of the hands; his speech was dysarthric and slurring. The Wassermann test of the blood was negative. Diagnosis: Progressive paralysis?

He would lie in bed for hours at a time, holding a newspaper, without reading it. He became worse gradually, failed to recognize his relatives, took little food, had dysphagia and involuntary urination and defecation. He developed pneumonia of the right lung and died.

Necropsy revealed pneumonia and mitral insufficiency. Section of the brain showed an easily enucleated tumor, the size of a hen's egg, in the left frontal lobe. The defect caused by the tumor involved the anterior two thirds of the first frontal convolution, the anterior half of the second frontal convolution, and had a total anteroposterior length of 4.2 cm. The convolutions of the anterior half of the left hemisphere were flattened, and the entire hemisphere seemed to be much broader than the right. This swelling began in the region of the anterior third of the tumor and increased posteriorly to such an extent that at the anterior central gyrus this hemisphere was about twice as large as its fellow on the opposite side; posteriorly this diminished to reach the normal configuration at the gyrus supramarginalis. Microscopic examination of the tumor showed it to be an endothelioma. The changes in the brain substance itself involved both the glia and nerve fibers. A dense glial layer surrounded the defect in the anterior and dorsal portions, outside of which was a very extensive etat criblé. This condition became accentuated posteriorly. There seemed to be no definite perivascular edema and no changes in the blood vessel walls. The meshes of neuroglia tissue were thickened. There was extreme parenchymatous degeneration, which in the vicinity of the tumor extended to a point just under the cortex, leaving the short association fibers intact. No changes were noted in the cortical gray matter. The density of the glial structures varied enormously, markedly rarified portions alternating with very dense ones. The corona radiata was moderately degenerated in the region of the anterior central gyrus. Only the posterior third of the left hemisphere was entirely normal. In the degenerated areas were numerous giant cells of glial origin, glia cells of moderate size, cells with a large amount of protoplasm, ameboid cells, spider cells and degenerative forms; gitter cells were present in abundance.

In general, it is to be noted that the glia increase, which must be looked on as a reaction to the dural tumor, in many places partakes of the characteristics of a tumor. The case in this respect resembles one reported by Merzbacher, in which a sarcoma, primary in the pia, was partially encapsulated on the surface abutting on the hemisphere by a secondary tumor, resembling a glioma. The doctrine of reactive gliosis has found few supporters. Pines, however, agrees with Merzbacher that here the glial structure must be looked on as a reaction product, secondary to the primary dural tumor. Obviously, then, it may be quite impossible to differentiate cell structures representing glial reactions from true diffuse glia tumors. Schmaus gives the following criterion for differentiating primary from secondary glial increase: In the latter instance the degenerated nerve fibers follow no definite configuration. Stroebe emphasizes the morphologic approach to normal glia tissue when a reactive gliosis is present. Accordingly, the presence of dividing cells and the variety of cell types would indicate true tumor formation. Bonome states that in secondary gliosis there is no increase in volume, and a disproportionate increase in fibers. All criteria, however, may fail when one tries to distinguish glial reaction from glial tumor.

Cohenheim-Ribbert's theory seems poorly adapted to the explanation of diffuse gliomas. Diffuse gliomas, however, show all transition stages from normal glia cells in the environment to undoubted tumor elements, so that the assumption presents itself that these tumor cells arise from normal tissue. The article is to be continued.

WOLTMAN, Rochester, Minn.

LOCALIZATION OF THE TONE SCALE WITHIN THE CORTICAL AUDITORY ZONE IN MAN. PFEIFER, Monatsch. f. Psychiat. u. Neurol. **50**:7 (July) 1921, and **50**:99 (Aug.) 1921.

Pfeifer presents an extensive discussion of the finer localization within the cortical auditory zone, with a review of the experimental, anatomic and clinical data. It has been fairly proved that the cortical center for hearing lies in the transverse gyrus of the temporal lobe. In a recent paper, the author has made a careful study of this zone and its projection system. In view of its variation in size and configuration, its overdevelopment in the brains of musical persons and its underdevelopment in deaf-mutes, he believes it to be not only a sense center, but a psychic center for musical perception.

The experimental work of Munk and of Larinow on dogs led both these authors to a belief in a finer localization of tones within the cortical auditory zone. Munk demonstrated that the center for lower tones lies posteriorly, the center for higher tones anteriorly. Larinow mapped out the zone still more completely, and attempted to transfer his observations to man. His attempt fell short because he was influenced by the theory of a dual auditory path, Wernicke having demonstrated that the sensory sense center for word perception lay in the posterior portion of the first temporal convolution, and Edgren having stated that the center for music perception lay in the temporal pole.

The author believes that the essential auditory center lies within the transverse convolution of the temporal lobe; that high tones are located in that portion of the gyrus which lies deep in the sylvian fissure, and that low tones are located in that part of the gyrus where it enters the convexity of the first temporal convolution. Owing to the variations in size and shape of the temporal transverse gyrus, these centers are subject to more or less displacement, which makes difficult the interpretation of clinical cases, especially as in most of these cases the finer pathologic studies have been incom-

plete. In the interpretation of clinical cases, he points to the further difficulty that the hearing tests are inadequate. Owing to the fact that most tuning forks have overtones, a patient for whom lower tones only are gone can still hear the low forks through their overtones. Interference with the lower tones leads to a much graver disturbance of musical appreciation than loss of the upper tones.

His conclusions are as follows:

1. Animal experiment, pathologic and anatomic studies of the brain indicate that within the cortical auditory area in man (transverse temporal gyrus) high tones and low tones have a separate localization.

2. The assumption is justified that high tones are situated mesially (root of the transverse gyrus in the depths of the sylvian fissure), and deep tones laterally (where the transverse gyrus enters the convexity of the first temporal gyrus).

3. The variable configuration of the transverse temporal gyrus in man makes it probable that the auditory zone is not simply a sense perception zone, but also a psychic center.

4. This variable configuration would seem to be the morphologic expression for individual variations in auditory perception, especially for music.

5. With total destruction of one auditory zone, the tone series can still be fully perceived by the opposite hemisphere.

6. In order to have the music sense intact, the auditory tract and auditory center on the left side must be intact. Total destruction of the auditory tract or auditory center on the left results in amusia, in spite of the fact that all tones can be perceived.

7. Considering the variability in the configuration and size of the auditory zone and its projection tracts, it is possible to explain many of the facts which led Edgren, Probst and Henschen to assume a music sense center in the region of the temporal pole on other grounds, cortical or subcortical amusia being due rather to involvement of the temporal transverse gyrus or its projection system.

8. Other authors have held that a remnant of the transverse temporal gyrus is sufficient to permit perception of all tones. In interpreting this one must remember two sources of error—the difficulty in testing for partial tone deafness and the difficulty in obtaining sources of tones free from overtones.

9. There is no evidence in the literature that the converse of the author's theory could hold, namely, that low tones are mesially situated, and high tones laterally.

SELLING, Portland, Ore.

COMPARATIVE STUDIES IN THE CHEMISTRY OF BLOOD AND CEREBROSPINAL FLUID. GRETE EGERER-SEHAM and C. E. NIXON, Arch. Int. Med. **28**:561 (Nov.) 1921.

The normal value for sugar in cerebrospinal fluid as determined by the investigators was 0.069 per cent. In cerebrospinal syphilis, tabes dorsalis, syphilis, hemiplegia, disseminated sclerosis, neurasthenia, brain tumor, arteriosclerosis and other non-neurologic diseases, the quantity of sugar was approximately normal. A slight increase in sugar was found in dementia paralytica but not sufficiently marked to be of value in diagnosis. In tuberculous meningitis there was a marked decrease in sugar. In diabetes the spinal fluid sugar was increased proportionately to the blood sugar.

The normal for creatinin in spinal fluid varied from 0.45 to 2.20 mg. for 100 c.c. of spinal fluid, but the ratio between pathologic blood and spinal fluid was not found to be sufficiently constant to be used clinically.

The normal for urea in spinal fluid was 9.87 mg. in 100 c.c. A slight increase of urea in the spinal fluid in cerebrospinal syphilis was found. In diseases of cerebrospinal involvement the average normal ratio of urea of normal spinal fluid and blood, 62.15 per cent., was slightly increased.

Under normal conditions the carbon dioxid carrying capacity of cerebrospinal fluid was somewhat lower than that of blood, while in acidosis it showed a tendency to become greater.

Spinal fluids were also examined for ferments. In two fluids among twenty-six the presence of lipase was merely suggested. Diastase, however, was found in all but two of the thirty cases; the highest diastase content was found in a case of spastic torticollis; low readings were obtained in meningitis. No regularity between the diastase content and sugar was noted. Trypsin was not found in the spinal fluids examined. The specific gravity of spinal fluid as determined by the investigators was 1.086 with no marked deviations even in severe neurologic disease.

With respect to syphilis, the authors conclude that cerebrospinal fluid shows no constant deviation from normal in sugar, creatinin, urea content, acid base equilibrium, enzymatic activity or in specific gravity.

VONDERAHE, Philadelphia.

A CASE OF BILATERAL CEREBELLAR ABSCESS WITH NO LOCALIZING SYMPTOMS. E. C. SPARR, Indian M. Gaz. **56**:12 (Dec.) 1921.

The author describes the case of an 8 year old girl, whose family and personal history were negative. Two months prior to admission to the hospital she was injured in her head and back. There was immediate pain along the spine and in the neck, and she slept for twenty-four hours. In ten days she was about her play as usual, but during the next month she became irritable and cried without cause, showed an aversion to sweets (having previously been fond of them), became seclusive and did not care to eat. Six days before admission she appeared feverish in the evening, had photophobia, supported her head with her hands and said her head was being crushed. She complained of a bitter taste in her mouth, and held her head and spine rigidly. On the day of admission she vomited soon after getting up and then became unconscious. On examination after admission she was unconscious and rigid, her pupils were dilated, there was a tendency to conjugate deviation to the right and external strabismus and slow nystagmoid movements were present. The tendon reflexes were all exaggerated; ankle clonus and Babinski sign were present. Spinal puncture gave a slightly turbid fluid with a few lymphocytes and polymorphonuclear cells, a trace of albumin but no sugar. The following day she had a convulsion. On the third day after admission the nystagmus was absent, the pupils were equal and contracted, and the external strabismus was not marked. On the fourth day athetoid movements of the fingers were present. Death occurred on the fifth day. The case was diagnosed as tuberculous meningitis. Necropsy revealed considerable increase in the cerebrospinal fluid. There were abscesses in both lobes of the cerebellum, the larger one being on the left side. Careful examination of

the lungs and other organs of the body gave no evidence of tuberculosis. Sparr remarks on the lack of symptoms suggesting cerebellar abscess and describes in detail those pointing to tuberculous meningitis.

POTTER, Mercer, Pa.

RECKLINGHAUSEN'S DISEASE: ITS RELATION TO THE ENDOCRINE SYSTEM. REPORT OF AN ILLUSTRATIVE CASE. OSCAR L. LEVIN, Arch. Dermat. & Syph. **4**:303 (Sept.) 1921.

The concept of Recklinghausen's disease is held by Levin to include, not only generalized neurofibromatosis, but incomplete forms, showing pigmentation, psychic, nervous and trophic disorders. A review of the literature bearing on the relationship of the disease to the endocrine glands is given. The first manifestation in Levin's case was a nevoid growth on the neck appearing a few months after birth. Freckles appeared at about the age of 4 and increased rapidly to yellow and brown spots of general distribution. At the age of 8 painful tumors of the scalp and vertebral region appeared and were removed. At the age of 16, other tumors were removed and the pigmented nevus of the neck became dark and covered with small growths. When the case was fully developed it presented the following symptoms, grouped by the author according to their suggested relationship to various endocrine glands:

"Suprarenals: Attacks of weakness, faintness and precordial pressure; hyperesthesia; muscular pains; poor vasomotor tone—diminished skin stroking reaction, low blood pressure; diminished metabolism—low blood sugar (0.07 per cent.) and urea nitrogen (8.0 per cent.); anorexia; nocturnal frequency of urination; insomnia; mental symptoms; abnormal hair growth—hypertrichosis of face, back, axillae, abdomen and between the eyebrows; obesity; dermatologic condition—pigmentation, fibroma, neurofibroma, nevus.

"Pituitary: Periodic frontal headaches; poor vasomotor tone; low blood sugar; nocturnal frequency of urination; insomnia; mental symptoms; low down growth of hair on the scalp; structural defects—short and obese, shape, size and asymmetry of face and head, prognathism, enlarged right ear, spacing and character of teeth, hyperextensibility of the fingers.

"Thyroid: Hyperesthesia, poor vasomotor tone; low urea nitrogen; thinned outer one third of eyebrows; obesity.

"Gonads: Menstrual symptoms; abnormal hair growth; obesity; dull lethargic mentality."

VONDERAHE, Philadelphia.

BISMUTH IN SYPHILIS. C. LEVADITI, Presse méd. **30**:633 (July 26) 1922.

This article is a review of recent literature on the use of bismuth preparations in syphilis, together with new case reports. The conclusion is reached that bismuth is a powerful and valuable antisyphilitic comparable with arsenic.

Some success had been noted with subcutaneous injections of bismuth salts, but the intramuscular route was the more satisfactory; the intravenous and oral routes were contraindicated.

Good results were obtained in primary, secondary and tertiary cases. No patients with dementia paralytica were benefited. A patient with acute syphilitic meningitis was symptomatically cured and became serologically negative in blood and fluid under intramuscular treatment. One observer had demonstrated bismuth in the cerebrospinal fluid during a course of the drug. No case was found in which the spirochete was resistant to bismuth, nor were any relapses observed after its use. It was effective when mercury and arsenic had failed.

HUDDLESON, New York.

MULTIPLE SCLEROSIS IN CHILDREN, WITH A REPORT OF THREE CASES. I. S. WECHSLER, Neurol. Bull. **3**:579 (Nov.-Dec.) 1921.

Wechsler reports three instances of multiple sclerosis in children, with the onset placed at the seventh, eighth, thirteenth and fourteenth years of life. The incidence of the disease in childhood is about 2 per cent.

STRECKER, Philadelphia.

THE DIAGNOSIS OF TUMORS OF THE CAUDA EQUINA, CONUS AND EPICONUS MEDULLARIS: A REPORT OF NINE CASES. HARRY L. PARKER, Am. J. M. Sc. **163**:342 (March) 1922.

Nine cases of tumor of the cauda equina, conus and epiconus medullaris are reported by Parker. The slower growing tumors were found to present clear cut signs, while the malignant types gave more diffuse signs. Tumors of the sacral canal were found to be well advanced before giving localizing signs. Of eight tumors, studied pathologically, two were encapsulated—one an endothelioma, the other a glioma; the remaining six showed a tendency to erode dura, bone and muscle. The salient features of the symptomatology of the cases studied are enumerated. Of these, pain is emphasized as being of particular diagnostic value. Pain is noted as occurring months and even years before any sign is established; it is intermittent at first, later constant and often relieved by walking or by rest in a sitting position. Next are mentioned tenderness of the spine, atrophic paresis of the lower limbs, perianal or saddle anesthesia associated with sphincteric disturbances, sensory changes varying from a slight loss of which the patient is unconscious to complete anesthesia of the lower extremities, and alteration of the tendon reflexes. Spinal puncture findings varied: a "dry tap" was obtained in three cases in which the spinal canal was choked by a large tumor; in one case xanthrochromic and massive coagulation phenomena were present; in two cases the Nonne test was positive; in one case, later found to be a glioma, the presence of a large number of pus cells led to a diagnosis of subdural abscess. Edema of the lower extremities was noted in two cases. When the symptoms and signs noted in the foregoing are present, the diagnosis is not difficult, but exact localization is often impossible because the degree of involvement frequently does not correspond to the symptomatology. Parker's ninth case is one of tumor of the pelvis pressing on the emerging trunks of the third, fourth, and fifth sacral and the coccygeal roots with their associated plexuses and ganglions, giving a clinical picture of caudal tumor; the case is cited as emphasizing the necessity of digital pelvic examination in all cases.

VONDERAHE, Philadelphia.

CONCERNING THE INFERIOR OLIVE. B. BROUWER and L. COENEN, J. f. Psychol. u. Neurol. **25**:2 (Aug.) 1919.

In this article the authors, after a short discussion of the newer literature on the inferior olive, take up two cases of their own in which pathologic changes in the olivary system were found. In the first case there was a small, fairly well localized lesion in the medioventral part of the cerebellum taking in the caudal half of the tonsil, a small part of the lobus cuneiformis and a small section of the lobus gracilis, while the vermis was entirely uninvolved. In this case there was found a considerable amount of degeneration in the opposite inferior olive, while the crossed pontile nuclei were normal. In

the second case a child had encephalocele in the occipital region which had been operated on and in which there was a unilateral atrophy of the cerebellum with a malformation of the vermis. These changes in the cerebellum caused pathologic changes in the inferior olives.

The authors deduct from these cases, in association with former studies and the facts which comparative anatomy teaches, the following: 1. The cells of the pontile nuclei send their axis cylinders to a part of the cerebellar hemisphere as the phylogenetically younger part of the olivary system. 2. The region of the tonsil and the bordering territory of the hemispheres must represent a rich projection system from the inferior olives. 3. The theory is substantiated that the para-olive and the frontal pole of the main olive are in anatomic relationship with the paleocerebellum, while the greatest part of the main olive is related to the neocerebellum. 4. The medioventral para-olive is in anatomic relation with the pars postrema cerebelli (pyramis, uvula, nodulus, flocculus and paraflocculus). 5. The medioventral accessory olive of water mammals is greatly enlarged, and at the same time the pars postrema cerebelli (especially the paraflocculus) is also greatly increased in size.

WINKELMAN, Philadelphia.

THE TREATMENT OF MENINGOCOCCUS MENINGITIS. KENNETH D. BLACKFAN, Med. **1**:139 (May) 1922.

This is an excellent analytical review of the clinical, bacteriologic and serologic history of meningococcus meningitis with special reference to treatment. Certain practical points stand out: In the premeningitic stage, the disease is a bacteremia although the organisms disappear from the blood soon after fixation occurs in the meninges. At this stage diagnosis, except during epidemics, is difficult, but treatment should be intravenous. There is some danger of precipitating meningeal fixation by lumbar puncture at this time, but the importance of early diagnosis outweighs this danger and can be made with certainty only by discovery of the organism in the spinal fluid. With the development of meningitis the serum must be administered intraspinally, and the intravenous administration may be discontinued. The balance of evidence is opposed to the view that the lateral ventricles are the primary seat of infection, and hence it is well to reserve intraventricular injection for very young children or for patients in whom the spinal fluid is too thick to run through the lumbar needle or is blocked off with adhesions. It should also be used in more severe cases in which there is danger of changes leading to hydrocephalus. The serum should be polyvalent and of good quality, the best test of potency at present available being the agglutination titer. No other mode of treatment has yet been proved satisfactory, although there may be use for vaccines in prophylaxis and as an adjunct to the serum in the treatment of the active disease.

Serum injections should be given at least as often as every twenty-four hours and must be controlled by studies of the spinal fluid, intracellularity of organisms and culturability. They should be discontinued when the fluid becomes clear and organisms can no longer be cultivated. If unduly prolonged, the serum may itself give rise to irritative effects, and if insufficient, the discontinuance may be followed by relapse. Sudden death following injection is probably due to too rapid introduction of the serum, which should be made by gravity very slowly.

SINGER, Chicago.

AN ENDOCRINAL FACTOR IN GENERAL PARESIS. THOMAS K. DAVIS, Am. J. M. Sc. **163**:425 (March) 1922.

Eighty-two unselected cases of paresis were examined by Davis with reference to the degree of trichosis. Of the patients possessing a low or average degree of trichosis, 29.5 per cent. died in less than two years, while 45 per cent. lived more than three years. Of the patients possessing more than average or excessive trichosis, 50 per cent. died in less than two years, while 30 per cent. lived more than three years. Assuming that "hypertrichosis is a symptom of suprarenal hyperplasia," Davis concludes that the course of general paresis varies in rapidity directly with the suprarenal strength of the individual.

VONDERAHE, Philadelphia.

THE PRACTICE OF PSYCHO-ANALYSIS IN A PUBLIC CLINIC. P. LEHRMAN, Neurol. Bull. **3**:362 (Nov.-Dec.) 1921.

In this paper Dr. Lehrman details his two years' experience with psychanalysis in the Vanderbilt Clinic. He treated twenty-seven patients (seven had anxiety and five conversion hysterias, four obsession neuroses, one psychic impotence, six conduct disorders, two cyclothymias, one dementia praecox and one paraphrenia) and apparently succeeded in clearing up the symptoms in seventeen. He also discusses the use of psychanalysis in a clinic for the purpose of diagnosis, claiming that in many instances analysis is the only method for arriving at a correct conclusion.

The author did not completely analyze all his cases; nor would psychanalysts concede that he carried out the accepted method. What he really did was to apply psychanalytic investigation to a number of difficult cases, and he succeeded, in more than a superficial way, in clearing up symptoms as well as diagnoses. In so far his paper is of value, showing, as it does, the possibility of psychanalysis in a clinic—a thing which psychanalysts have hitherto declared to be impracticable.

WECHSLER, New York.

NEURINOMA IN RECKLINGHAUSEN'S DISEASE. EUGEN KIRCH, Ztschr. f. d. ges. Neurol. u. Psychiat., No. 74: 379, 1922.

Multiple neurofibromatosis has been investigated a number of times, and the occurrence of nerve fibers in the characteristic tumors has been almost universally denied; but in this case Kirch was able to find them in large numbers. The arrangement of the cells in bundles, so-called phalangiform, also agreed with the description of the true neurinoma according to Verocay. There were no tumors of the central nervous system or of the smaller peripheral nerves, but there were many in the sympathetic plexuses in the walls of the intestine. Besides the nerve fibers, the tumors contained also isolated myelin sheaths, but ganglion cells were almost or entirely lacking. Kirch believes these axis cylinders to be the results of regenerative budding of preexisting axis cylinders. The case, he says, is a textbook picture of multiple neurinoma.

FREEMAN, Philadelphia.

MENINGITIS OSSIFICANS, COMPRESSION MYELITIS, OPERATION, RECOVERY. L. PUSSEP, Ztschr. f. d. ges. Neurol. u. Psychiat., No. 74: 415, 1922.

After a fall the patient noted stiffness and weakness of the legs, which increased gradually and in the course of a year resulted in subtotal paralysis.

Level symptoms were indefinite, but at operation in the thoracic region a bony plaque was found in the arachnoid. Eighteen centimeters of the cord was then explored, and six similar plaques found, each about 10 mm. in diameter and 3 mm. in depth. They pressed into and distorted the cord but were easily lifted out, and the deformity soon disappeared. The patient made an uneventful recovery and returned to duty.

FREEMAN, Philadelphia.

LATENT TIME OF REFLEXES. HARRY SCHÄFFER, Ztschr. f. d. ges. Neurol. u. Psychiat., No. 74: 605, 1922.

Schäffer, working in Minkowski's laboratory, has devised a new method of determining the latent period of reflexes through the use of the electromyogram. The results are accurate and easily measured. In discussing the question of the nature of the knee reflex, the author says it is indeed a true reflex, and that the shortness of its latent period is due to the simplicity of the central nervous mechanism.

FREEMAN, Philadelphia.

OBSERVATIONS ON THE ETIOLOGY AND PATHOLOGY OF CHOREA MINOR. LOUIS C. SCHROEDER, J. A. M. A. **79**:181 (July 15) 1922.

The author reports a case of chorea minor in a 20 months old white baby. Blood cultures were negative, but necropsy showed that both heart and pericardium were involved, and microscopic examination of the heart muscle revealed typical Aschoff nodules. These nodules are looked on as strong presumptive evidence of a preceding rheumatic infection.

The author also notes that advances in the study of the physiology of the basal ganglions make it seem likely that the chief pathologic changes in chorea minor occur there.

NIXON, Minneapolis.

CONTAGIOUSNESS OF EPIDEMIC ENCEPHALITIS. GEORG STIEFLER, Ztschr. f. d. ges. Neurol. u. Psychiat., No. 74: 396, 1922.

From eight personal observations and comparison with cases previously reported, Stiefler concludes that epidemic encephalitis is transmissible from person to person by contact and even through the intermediation of a healthy third person. The contagiousness, however, is slight, less than that of infantile paralysis.

FREEMAN, Philadelphia.

ANATOMIC STUDIES OF THE VISCERA IN DEMENTIA PRAECOX. F. WITTE, Ztschr. f. d. ges. Neurol. u. Psychiat., No. 72: 164, 1922.

In the majority of cases there is a decrease in the lipoids of the suprarenals, as is the case in chronic infections such as tuberculosis and some acute septic processes. In the nervous system and bodily organs, no marked alterations from normal were appreciable.

FREEMAN, Philadelphia.

LATENT NEUROSYPHILIS IN EIGHT PER CENT. OF MEDICAL PATIENTS IGNORED OWING TO NEGLECT OF LUMBAR PUNCTURE. H. GRAY, Am. J. M. Sc. **165**:384 (March) 1922.

In sixty-two medical cases, all supposedly nonvenereal, Gray found eight syphilitic patients, only two of whom knew of their disease. Five of these had involvement of the central nervous system. In view of such an incidence

Gray states that a lumbar puncture should be performed on every syphilitic patient, and repeated annually, if positive, until it has been negative for one year; and thereafter every two years, to exclude recurrence.

VONDERAHE, Philadelphia.

A CASE OF TUMOR OF THE SPLENIUM OF THE CORPUS CALLOSUM.

G. GUILLANI, Rev. Neurol. **29**:23 (Jan.) 1922.

The case here reported may be summarized as follows: The patient (a man, aged 52) with apparently negative family and early personal history, entered Guillani's service with a complaint of failing memory, headaches and vertigo of seven months' duration. On examination the patellar and Achilles reflexes were found to be absent, as well as the plantar, abdominal and cremasteric responses. There was marked urinary retention. The reflexes over the brachial field were much diminished, and there was a constant coarse tremor of the right arm. The Kernig sign was present, and the pupils were unequal with greatly diminished reaction to light and accommodation. Vision was much impaired and there was evidence of spontaneous nystagmus; but, owing to poor cooperation, no examination of the fundi could be made. Apraxia was characteristically present. On lumbar puncture there were found 32 cells, hypertension and somewhat increased solids with a slightly positive Wassermann and a negative colloidal gold reaction. Mentally there was evident a certain general deterioration with associated alternating agitation and depression and periods of marked hebetude and confusion.

When seen two months later the patient's mental state was one of practically continuous torpor unassociated apparently with any further neurologic change. The spinal fluid at this time showed much increased pressure, 7 cells and a greatly increased amount of solids with again weakly positive Wassermann and negative colloidal gold reactions. Death occurred soon afterward following a period of stupor.

Necropsy revealed a cystic hemorrhagic glioma, apparently primary in the splenium of the corpus callosum, but secondarily involving the internal lateral ventricular wall, the tapetal white matter, the posterior thalamus and thalamic radiations, the inferior longitudinal bundle bilaterally, the white matter of the first limbic convolution and the thalamic radiations, the forceps inferiorly and the cingulum posteriorly.

On the basis of the features presented by this case and others reported in the literature Guillani feels that a definite clinical syndrome may be validly assumed thus as regards callosal tumors:

1. Evidence of increased intracranial pressure.
2. Early and progressive psychic change, that is, deterioration and confusion with alternating agitational and torporous periods strongly suggestive on the whole of the paretic reaction.
3. Frequent motor disorder and apraxia unattended by aphasia and basal cranial palsies.

RAPHAEL, Ann Arbor.

THE TREATMENT OF ANTENATAL AND CONGENITAL SYPHILIS.

JOHN A. FORDYCE and ISADORE ROSEN, Arch. Dermat. & Syph. **5**:1 (Jan.) 1922.

The authors recommend a course of treatment for syphilitic women during pregnancy consisting of from six to eight doses of arsphenamin or neoarsphenamin, with mercuric chlorid or mercuric salicylate, 1 grain, once a week

for twelve or fifteen injections. Many infants born of treated mothers are without clinical or serologic evidence of infection, although many of the mothers have postive findings. A hundred and forty infants, with negative reactions but born of mothers with strongly positive findings, were observed for a year and a half. Forty-eight of the mothers received no antenatal treatment; twenty-four received internal treatment only; sixty-one received partial treatment with arsphenamin and mercury; and only six received prolonged treatment with these drugs.

In one case a woman with a negative blood Wassermann reaction but a positive spinal fluid Wassermann reaction gave birth to a child in whom the blood from the umbilical cord was strongly positive although no clinical evidence of the disease was noted in the infant. In another case, a man, aged 35, who acquired syphilis at the age of 18, was treated by inunction for two years and was married at the age of 22; four children were born, all negative clinically and serologically; the wife gave a negative blood and spinal fluid Wassermann reaction, while the husband had an advanced tabetic condition with a strongly positive blood and spinal fluid.

The authors' treatment for congenital syphilis consists of intramuscular injections of neo-arsphenamin in the following dosage: 0.1 gm. for infants from 2 to 12 weeks old; 0.15 gm., from 3 to 9 months; 0.2 gm., from 1 to 2 years, and 0.25 to 0.3 gm. for children 3 years old. Ten or twelve weekly injections of mercuric chlorid are given in the following dosage: 1/10 grain for children from 2 weeks to 6 months old; 1/8 grain from 6 months to 1 year; 1/7 grain from 1 to 2 years; 1/5 grain from 2 to 3 years and 1/4 grain for those more than 3 years old. In eighty-eight children with strongly positive Wassermann reactions, twenty gave clinical manifestations which yielded promptly to the treatment. Fourteen of forty-seven treated patients became negative serologically.

VONDERAHE, Philadelphia.

PSEUDOTUMOR SYNDROME DEPENDENT ON ACUTE SWELLING OF THE BRAIN. C. I. URECHIA, Rev. Neurol. **27**:1185 (Dec.) 1920.

Comment is made on the etiologic and pathologic obscurity still prevailing as regards the so termed pseudotumor syndrome. Particular attention is directed to the possible etiologic rôle in this regard of the acute swelling of the brain (akute Hirnschwellung). The author feels that this is brought about through ameboid changes in the neuroglia cells with consequent increase in gross brain volume.

An illustrative protocol is cited in which the patient (a woman, aged 34) presented herself at the author's clinic with a history of hebetude, general weakness and severe headaches of recent onset and, on neurologic examination, sluggish unequal pupils, exaggerated deep and superficial reflexes, patellar cloni and occasional Babinski reaction. Examination of the spinal fluid was negative except for an indication of increased solids. Death occurred three days later, following a period of stupor, with the neurologic examination indicating profound general motor paresis, strongly positive bilateral Babinski response, patellar and ankle cloni, incontinence, extremely severe headache, slight rise in temperature and, as regards the fundi, evidence of papillitis and optic atrophy bilaterally. Postmortem examination was grossly negative except for an indication of slight congestion of the meninges. Microscopic examination showed profound chromatolytic changes affecting cells throughout the cortex. The neuroglia cells showed in addition marked ameboid change, particularly intense

periventricularly and much less so in the gray matter itself. This change was characterized by swelling of the cells, process loss, hyperchromatic nuclei with halo formation, cytoplasmic, hyperchromism and evidence of severe granular, fatty, vacuolar and cystic degeneration.

RAPHAEL, Ann Arbor.

FRACTURE-DISLOCATION OF THE SPINE TREATED BY FUSION.

RUSSELL A. HIBBS, Arch. Surg. **4**:598 (May) 1922.

From a study of twenty-two cases of fracture dislocation of the spine, Hibbs concludes that many injuries of this type are unrecognized. A very thorough roentgen-ray study with lateral, anteroposterior and stereoscopic plates is necessary to confirm the diagnosis and justify operative interference. In spite of the thorough roentgenographic examination in many cases the surgical exposure revealed more extensive damage than the roentgen ray suggested. In his series of cases the fracture dislocation was located once in the cervical region, twice in the dorsal and dorsolumbar and seventeen times in the lumbar region. Of the last named, eleven were located in the fifth lumbar vertebra. He emphasizes the fact that the lumbar spine is most susceptible to fracture and that the fifth lumbar vertebra is the one most frequently injured.

The chief symptoms were deformities of the spine, protective postures, and a sense of weakness in the back. In the cervical case pain in the shoulders, neck and arms, was the chief symptom.

Patients with dorsolumbar and lumbar cases complained of pain in the legs resembling sciatica. Cord or nerve root symptoms were noted in only one case. In all cases the condition was crippling. In only three cases, all of recent fracture, was the diagnosis made; in the other nineteen cases, fractures had occurred from one to twenty-five years previously. An injury in childhood may apparently be of trivial importance yet show severe symptoms in adult life.

The symptoms are caused by the mobility of these altered joints and ununited fractures. Elimination of motion is essential to complete and permanent relief. Operative interference producing a fusion of the articulating bones is the treatment recommended. The surgical technic is described in detail.

GRANT, Philadelphia.

HYPOTHYROIDISM WITH UNUSUAL SKIN MANIFESTATIONS.

HARVEY P. TOWLE and E. LAWRENCE OLIVER, Arch. Dermat. & Syph. **5**:88 (Jan.) 1922.

A child, 3 years old, backward in its development, had coarse, dry hair; the hands and fingers were short and pudgy. The Wassermann and von Pirquet reactions were negative, and there was a deficiency of 19 per cent. in the basal metabolism. The skin, with the exception of the face, palms, soles and scattered areas on the legs, was reddened and swollen, and there were groups of eruptive lesions. The primary lesion appeared to be a turbid vesicle which grew peripherally and rapidly undermined and loosened the epidermis as it spread; coalesence took place and the process continued until the disease became universal. Smears and cultures from the lesions were negative in every instance. Various applications to the skin were tried, but with no effect; within two weeks, however, using thyroid extract, the child's mentality improved markedly and the skin was almost cleared.

VONDERAHE, Philadelphia.

INFLUENCE OF HEAD AND BODY POSTURE ON SPINAL FLUID PRESSURE. N. Zylberlast-Zand, Rev. Neurol. **28**:1217 (Dec.) 1921.

The author calls attention to the importance of the hydrostatic factor in the determination of spinal fluid pressure, feeling that its valence in this regard is distinctly greater than 24 per cent. as reported by Pfaundler, 68 per cent. being attributed to blood pressure and 8 per cent. to meningeal elasticity. Thus, Zand found that the pressure in normal persons ranges from 200 to 350 mm. in the sitting position, and in the lateral decubitus from 10 to 100, while in subjects presenting meningeal pathology, a range of from 150 to 200 mm. was determined in the lateral decubitus and a maximum of 400 in the siting position. Frequent inability to obtain fluid on puncture in the reclining position, while probably dependent in part on primary pressure fall in this posture, seems to be due chiefly to mechanical interference from the cauda equina. The author found, through observations on cadaver material, that in the reclining position the cauda equina showed a marked tendency to adhere to the posterior canal wall and in this manner to obstruct free circulation of the spinal fluid. It is of interest also that a rise in fluid pressure was consistently noted to follow head flexion in the reclining position (from 20 to 120 mm.), while in the sitting position this maneuver was productive of definite pressure decrease (— 50). The author regards this as due most probably, in the first case, to venous stasis dependent on disturbance of venous return-flow by flexion at the neck, in the second case (in the sitting posture) being mechanically less effective due to stress dissipation over the dorsal vertebrae.

Raphael, Ann Arbor.

HEMORRHAGIC MENINGO-ENCEPHALITIS IN ANTHRAX. B. Shanks, Indian M. Gaz. **56**:11 (Nov.) 1921.

This is a case report with gross necropsy findings. A man, aged 20, while suffering with a small anthrax pustule on the right malar prominence developed a temperature of 104 F., with signs of meningeal irritation. Examination of the spinal fluid showed the presence of blood, polymorphonuclear leukocytes, lymphocytes, and many typical bacilli. A blood culture was positive for anthrax bacilli. Death occurred in less than twenty-four hours. At necropsy the dura was tense and bulging; there was evidence of hemorrhage into all parts of the subarachnoid space and pinpoint hemorrhages into the cerebral cortex. There were hemorrhages into the small intestine and gross softening of the left parotid gland. Postmortem cultures from the brain and spleen gave a pure growth of anthrax bacilli. Guinea-pig inoculations resulted in death in twenty hours. The author summarizes briefly forty-five previously reported cases and calls attention to the fact that in only 50 per cent. of the cases in which the location of the primary lesion was recorded were they in the head. He concludes that meningo-encephalitis in anthrax is the result of a bacteremia.

Potter, Mercer, Pa.

A STUDY OF THE INCIDENCE OF HEREDITARY SYPHILIS. P. C. Jeans and J. V. Cooke, Am. J. Dis. Child. **22**:402 (Oct.) 1921.

The authors studied the placental changes and the Wassermann reaction on the cord blood in 2,030 infants. The series included pregnant women delivered in eight months in four hospitals caring for those able to pay the fees charged private patients, and the charity cases cared for in two hospitals and two outpatient obstetrical clinics. The blood of 389 infants was

examined, and it was found that the proportion of cases of hereditary syphilis that could be diagnosed by the placental examination alone was 27 per cent., while from the Wassermann reaction on the cord blood 63.6 per cent. of the cases could be recognized. Using this method on the entire series, the incidence of hereditary syphilis in the colored race was found to be 15 per cent., 1.8 per cent. in the poor of the white race and less than 1 per cent. in the well-to-do classes.

VONDERAHE, Philadelphia.

AN EXPERIMENTAL STUDY OF METHODS FOR BRIDGING NERVE DEFECTS, WITH A DESCRIPTION OF A NEW METHOD OF AUTOTRANSPLANT (AUTO-AUTOTRANSPLANT). ERNEST SACHS and JULIAN Y. MALONE, Arch. Surg. **5**:314 (Sept.) 1922.

Experimental results with three methods of bridging defects in the continuity of nerve trunks are described: (1) the direct, an anastomosis of the central and peripheral ends of an injured nerve into longitudinal incisions in a neighboring normal nerve; (2) anastomosis of the central and peripheral end of an injured nerve to flaps cut in the same quadrant of a normal nerve; (3) an auto-autotransplant of the central end of the injured nerve, the removed segment being just long enough to bridge the defect between the severed nerve ends.

The ordinary technic of nerve suture was carried out with great care, hemostasis being made as complete as possible. A close approximation of the nerve tissue in the cut ends was obtained, and sutures were passed only through the perineural sheaths.

The results of these experiments show that the axis cylinders of severed nerves will grow down or between medullary tubes of a functioning nerve without interfering with the power of such nerves to transmit impulses or losing their own ability to function. Nerve tissue seems to form unquestionably the best pathway for the regeneration of nerve tissue. The introduction of any foreign material in an attempt to form a pathway down which a nerve may grow is of doubtful value. The method by which the anastomosis is made—lateral, direct, lateral into flaps cut in the nerve trunk, or by auto-autotransplant, seemed to make little difference in the result. All were effective. The lateral direct transplant seemed the most satisfactory. But with a careful suture, the avoidance of scar tissue, and the introduction of nerve tissue as a pathway for the regeneration of the nerve, the chances of a good functional recovery seemed greatly improved.

GRANT, Philadelphia.

OBSERVATIONS ON INFANTILE TETANY. B. KRAMER, F. F. TISDALL and J. HOWLAND, Am. J. Dis. Child. **22**:431 (Nov.) 1921.

The authors emphasize the constancy of a lowered concentration of calcium in the serum of infants affected with tetany and bring forward evidence to support the view that calcium deficiency is the important factor in increasing neuromuscular irritability. In determining the amount of inorganic phosphorus in blood serum in tetany a marked variation was noted, and in about one half of the cases the concentration was found to be normal or slightly above normal; they conclude accordingly that an increase in inorganic phosphorus is not responsible for the characteristic symptomatology of the disease. The concentration of sodium and magnesium was found to be essentially normal and that of potassium slightly increased, so that the increase in the ratio of sodium and potassium to calcium and magnesium is almost wholly due to a decrease in the concentration of calcium.

VONDERAHE, Philadelphia.

NEUROLOGIC SIGNIFICANCE OF THE "NONNE REACTIONS." O. Gallotti and J. Schettino, Rev. Neurol. **27**:1085 (Nov.) 1920.

The authors direct attention to the importance diagnostically of the "Nonne Reactions" so-called in neurosyphilis, namely: (1) phase 1 of the Nonne-Apelt test for globulin; (2) spinal fluid pleocytosis (6+), and (3) the Wassermann reaction on the blood and spinal fluid.

Phase 1 of the Nonne-Apelt reaction has been found 95 per cent. positive (Nonne) in general paresis, 90 per cent. in tabes and 100 per cent. in meningovascular syphilis.

Pleocytosis (Vianna and Moses) occurs to the extent of 100 per cent. in general paresis, 100 per cent. in tabes and 64 per cent. in meningovascular syphilis.

The blood Wassermann reaction has been found 100 per cent. positive (Nonne) in general paresis, from 60 to 70 per cent. in tabes, and from 80 to 90 per cent. in cerebrospinal syphilis, while the Wassermann reaction on the spinal fluid has been 93 per cent. positive (Vianna and Moses) in general paresis, 25 per cent. in tabes and 10 per cent. in meningovascular syphilis and, respectively, 100 per cent., 100 per cent. and 96.2 per cent., when utilizing the Hauptmann-Hoessli method, that is, use of increasing amounts of spinal fluid in arithmetical progression.

It is of interest that no place is given the colloidal gold and mastic reactions in this serodiagnostic heirarchy.

Raphael, Ann Arbor.

THE TREATMENT OF SYPHILIS BY MERCURY INHALATIONS: HISTORY, METHOD AND RESULTS. H. N. Cole, A. J. Gericke and Torald Sollmann, Arch. Dermat. & Syph. **5**:18 (Jan.) 1922.

The authors review the history of mercury inhalations, describe their own method and record their observations. In five patients with active syphilitic lesions calomel was used in doses of 3 to 80 mg., totaling 225 mg. in two weeks. The weighed amount of calomel was heated in a tube, and the vapor inhaled by the patient as it was formed. In all instances but one bronchial irritation, salivation and tenderness or edema of the gum occurred; but there was no renal disturbance and no therapeutic response. In six cases metallic mercury, vaporized in a similar tube, was used, the patient receiving from 225 mg. in two weeks to 750 mg. in three weeks. No systemic or local effects were noted. There was no salivation, except in one doubtful case, and no evidence of improvement in the disease.

Vonderahe, Philadelphia.

EARLY DIAGNOSIS IN GENERAL PARESIS AND TABES. G. R. Lafora, Rev. Neurol. **27**:1190 (Dec.) 1920.

Lafora emphasizes the paramount importance of early diagnosis in general paresis and tabes, particularly from the standpoint of actual therapeutic prospect, feeling that unless these situations are detected extremely early in their development—in their preclinical phase, so to speak—little essential improvement serologically or pathologically is to be anticipated, even under the most intensive specific therapy. Attention is directed especially to early pathognomonic changes occurring in the spinal fluid in such cases, particularly in those of the paretic order. Illustrative protocols are cited in which there is reported pleocytosis and increased solids, with definitely positive Wassermann

and colloidal gold reactions. There was complete absence of psychiatric and neurologic symptomatology, with the exception of pupillary inequality and partial iridoplegia and negative or weakly positive blood Wassermann reactions.

RAPHAEL, Ann Arbor.

A CASE OF TUBERCULOUS MENINGITIS WITH A DRY SPINAL SUBARACHNOID SPACE DUE TO DIFFUSE TUBERCLE INFILTRATION OF THE SPINAL MENINGES. JOSEPH C. REGAN and G. W. HOLMES CHENEY, Am. J. Dis. Child. **22**:516 (Nov.) 1921.

Seven days before the termination of the illness a lumbar puncture was made and 12 c.c. of fluid were removed. The following day another puncture was made and 6 c.c. of fluid were obtained. Subsequent punctures on the same day resulted in only a few drops of fluid or none. After this ventricular punctures were made. The gross pathologic condition in the cord was essentially an obliteration of the subarachnoid space as a result of intense tubercle formation; the brain showed similar marked tubercle formation over the superior surface, the base and the walls of the lateral ventricles, including the choroid plexus. Microscopic examination confirmed the diagnosis of tuberculosis.

VONDERAHE, Philadelphia.

THE VALUE OF VENTRICULOGRAMS IN THE LOCALIZATION OF INTRACRANIAL LESIONS: THREE CASES OF OBSTRUCTIVE HYDROCEPHALUS AND ONE OF BRAIN TUMOR. E. B. TOWNE, Arch. Surg. **5**:144 (July) 1922.

Towne reports three cases of obstructive hydrocephalus and one of brain tumor in which the diagnosis was made by means of the ventriculogram. One of the cases of hydrocephalus was of the rare type in which one lateral ventricle was displaced and the hydrocephalus caused by a tumor (glioma), blocking the foramen of Monroe on one side. The other two cases of hydrocephalus were apparently due to blockage from unknown cause, probably meningitis about the basal cistern. In the case of the brain tumor a localization in one or the other frontal lobe was made from the neurologic signs, and the ventriculogram determined in which hemisphere the lesion was located. The technic recommended by Dandy was used throughout.

GRANT, Philadelphia.

HYPOPHYSEAL OBESITY AS A RESIDUAL SYMPTOM IN A CASE OF LETHARGIC ENCEPHALITIS. STIEFLER, Monatsch. f. Psychiat. u. Neurol. **50**:123 (Aug.) 1921.

A girl, aged 13, suffered a typical attack of lethargic encephalitis in February, 1920. Convalescence occurred after three months; after five months she was fairly well, except for a little dulness and apathy. After seven months she began to note a gain in weight, polyuria, polydipsia, loss of hair and a tendency to sweat on slight exertion. Growth was somewhat slowed. After fourteen months the condition was about the same except that her weight had increased further, and the polyuria was somewhat more marked.

The author attributes the syndrome to hypophyseal dystrophy. He is unable to determine the actual site of the lesion, whether it is in the hypophysis itself or in the adjacent basal structures of the brain.

SELLING, Portland, Ore.

DIAGNOSIS AND TREATMENT OF INTRATHECAL TUMORS OF THE SPINAL CORD. W. Thorburn, Brit. M. J. **1**:3185 (Jan. 14) 1922.

The author describes a typical case of tumor on the left anterolateral aspect of the spinal cord, at the level of the eighth dorsal vertebra, and a case which he calls "a blurred image of an intrathecal tumor" (probably a localized infection of the meninges producing an obstruction of the spinal subarachnoid space with pressure on the cord). Thorburn urges the careful examination of patients with intrathecal tumors of the spinal cord and the early removal of these tumors, as this increases the probability of complete recovery. He further urges the early exploration of all doubtful cases of transverse lesion of the spinal cord. He is of the opinion that many cases of transverse myelitis may be arrested or cured by incision and drainage of the dura mater, since such myelitis is often due to infection spreading along the spinal nerves and crossing the intradural space before the cord itself is attacked.

Potter, Mercer, Pa.

AN ANALYSIS OF NINETY CASES OF FUNCTIONAL DISEASE IN SOLDIERS. John M. Swan, Arch. Int. Med. **28**:586 (Nov.) 1921.

In Swan's patients, 65.5 per cent. had been recruited from occupations of a sedentary type and 51.7 per cent. had a family history of nervous disease. In 89.6 per cent. of the cases there was a history of subacute or chronic infections while in 87 per cent. there were present complications of an infective character, such as tonsillitis, nasopharyngitis, sinusitis and infected teeth; and in about 50 per cent. of these cases there were physical signs pointing to a disturbance of the endocrine system.

Vonderahe, Philadelphia.

KYPHOSCOLIOSIS AS AN EARLY CLINICAL SIGN IN SYRINGOMYELIA. C. Foix and E. Fatou, Rev. Neurol. **29**:28 (Jan.) 1922.

Foix and Fatou cite in careful detail two cases in which there had existed marked dorsal kyphoscoliosis for many years (thirty and fifty) prior to the development clinically of typical sypringomyelic features. It is of especial interest that in these cases which seem to have been of predominantly unilateral type, the side most severely affected was that toward which the convexity of the spinal deflection was directed, dependent apparently on diminished vector efficiency as regards the vertebral musculature on the affected side. The authors believe that this initial kyphoscoliosis was essentially a manifestation of early abortive sypringomyelia, remaining latent over a long subsequent period with final recrudescence in typical form. This confirms, in a certain measure, the claim advanced some years ago by Leyden as to the frequent congenital nature of syringomyelic change.

Raphael, Ann Arbor.

CONTRIBUTION TO THE STUDY OF LATE HEREDITARY SYPHILIS. Julius Schuster, Monatsch. f. Psychiat. u. Neurol. **50**:152 (Sept.) 1921.

The author reports a case of late hereditary syphilis with necropsy. In addition to the ordinary histologic changes, interesting lesions were found in the Purkinje cells of the cerebellum. Throughout the entire cerebellum these cells were greatly swollen; their dendrites showed patchy balloon-like enlargements, and both cell body and dendrite were packed with a fine brown

pigment. This picture is similar to that described by Straussler, Schobb and others in the group of nonsyphilitic heredo-degenerative diseases, on the basis of which Schaffer propounded his hyaloplasma theory of the origin of the heredodegenerations. The author's case shows that similar changes may occur in hereditary syphilis.

SELLING, Portland, Ore.

CASE OF SPINAL CARIES AND COMPRESSION MYELITIS, LATERAL SCLEROSIS. J. MOORE, Dublin J. M. Sc. **4**:23 (Jan.) 1922.

The author reports the case of a man, aged 41, who suffered with signs of pulmonary tuberculosis at about the age of 22. At 34 he had a tuberculous testicle removed, and at 40 he noted weakness of the lower extremities, followed by loss of power in both lower limbs. Examination showed spasticity of both lower limbs, greater on the left. There was no loss of deep, epicritic or protopathic sensibility. Superficial reflexes (abdominal, cremasteric and epigastric) were exaggerated and later lost. Bone graft to the spine was done after two months' rest in bed. Nine months after operation he was practically restored except that the left interossei did not respond well to faradic stimulation, although the galvanic response was normal.

POTTER, Mercer, Pa.

CONGENITAL GOITER. REPORT OF FOUR CASES. WILLIAM B. PORTER and R. A. VONDERLEHR, Am. J. Dis. Child **22**:477 (Nov.) 1921.

The four cases reported occurred in male children. In no instance were there any toxic thyroid symptoms or pressure symptoms. The goiters in all cases were enlarged symmetrically, the most prominent part being the isthmus. They were soft in consistency and suggestive of goiters of the colloid type. A noteworthy finding was the presence of the ova of *Ascaris lumbricoides* in the feces in all four cases

VONDERAHE, Philadelphia.

A CASE OF SYRINGOMYELIA. W. S. ROBERTSON, Lancet **201**:1272 (Dec. 17) 1921.

The author reports a typical case of syringomyelia, beginning at the twenty-sixth year, which was progressive for a period of four years, two years of which time the patient was bedridden. During the fifth year she began to improve and after about one year was able to do all her own housework. Examination ten years later showed only increased knee reflexes and patches of disassociated anesthesia on the plantar surface of the feet. Robertson remarks on the rare occurrence of such an almost complete recovery in syringomyelia.

POTTER, Mercer, Pa.

Book Reviews

KATATONIE UNTER DEM BILDE DER HYSTERIE UND PSYCHOPATHIE. By M. URSTEIN. Paper. Pp. 465. Berlin: S. Karger, 1922.

This is the final volume of the series of clinical studies on katatonia by this author, the first of which appeared in 1909. As in the earlier volumes, the material is derived from studies and observations of patients over a long series of years. The records of twenty cases are given in great detail; in eighteen of them hysterical or psychopathic traits, sometimes mixed with manic-depressive features, have been prominent and even predominant for many years before the appearance of katatonia and the final characteristic dementia; in one the katatonic symptoms occurred in connection with a paretic, and in the last with a polyneuritic picture. Following the case records, each accompanied by a critical summary, is an excellent, concise discussion of the general symptomatology, nature, course and outcome, prognosis and diagnosis.

Urstein reiterates his previously published conviction that katatonia is a definite disease entity, the essence of which is some "endocrine, anaphylactic" disturbance," and insists that even when the earlier picture has been dominated by hysterical or psychopathic features "this should not be regarded as evidence that the katatonia is an addition, but rather that the condition was katatonic from the start." Further, "While the presence of katatonic features indicates the existence of this disorder, yet their absence does not prove the contrary."

Whatever view be taken of this concept, there can be no question as to the great debt which psychiatry owes to Urstein for the painstaking exactitude of record and keen-sighted analysis of clinical facts which form the basis of the present and preceding volumes. He illustrates admirably the need for such extended observations by giving the further course and outcome of cases which have previously been published by others.

Urstein recognizes several varieties of onset and course: 1. The primary manifestations, often for long periods, are those of hysteria, psychopathic personality, neurasthenia, hypochondria or compulsion neurosis, the katatonic symptoms being added gradually and leading to a typical end-state. 2. Phobias or psychopathic phenomena appear simultaneously with katatonic symptoms. 3. Manic-depressive episodes, mixed with which are katatonic symptoms, with perhaps temporary recovery are followed by hysterical or degenerative features and finally typical katatonia. 4. Rarely, the early symptoms are those of acute katatonia with the later addition of pronounced hysterical or degenerative traits. He maintains, and his records go far to prove, that the evolution from the psychoneurotic or psychopathic picture to final katatonia can be foreseen if careful attention is paid to the presence of evidence of real intrapsychic splitting and disharmonies between thoughts, emotional expressions and acts.

The outcome in all forms is much the same except in degree of dementia which varies greatly with the quality of the material of which the individual is built, the environment and the treatment. Where these are favorable, the final dementia may be long postponed and less severe. Once katatonic symptoms become marked, remissions are rare. When the early stage is dominated by hysterical features, the course is usually progressive; and when they appear late, the prognosis is more unfavorable.

EPILEPSIE UND MANISCH-DEPRESSIVES IRRESEIN. Von Dr. Hans Krisch. Abhandlungen aus der Neurologie, Psychiatrie, Psychologie und ihren Grenzgebieten. No. 18. Price, 36 marks. Pp. 107. Berlin: S. Karger.

That there is no uniform "epileptic character," that psychic epilepsy cannot be demonstrated solely on the basis of mental symptoms, and that essential epilepsy is a tenable diagnosis, are the main conclusions derived by Krisch from his studies of 200 cases of epilepsy. Of these, 140 were of the "genuine" type; the others were largely symptomatic. In a third to a fourth of the essential types a direct heredity was traceable, besides numerous instances of psychopathic manifestations in the collateral branches. The predominant fact in epilepsy is the inherited constitutional factor. Krisch presents detailed case histories of those cases showing interesting affective disorders and compares them with the symptoms of manic-depressive psychosis. The differences are attributed largely to the obscuring of consciousness in epilepsy which prevents the development of the "nuances" familiar in the affect syndromes of the "manic-depressive." The epileptic patient in his depression shows a less coherent picture, is more inclined to be accusatory than self-derogatory, is likely to commit suicide, fight or run, and is abrupt and brief in the onset and termination of his emotional disturbance. During the disorder he contrasts with the manic-depressive in his violence, often in a changed expression of his eye and in his stertorous breathing. Krisch finds that the affect symptoms are in themselves not sufficiently differentiated from manic-depressive reactions to be diagnostic. Unless there is a history of preceding convulsions or a definite obscuring of consciousness during the emotional disorder, it cannot be diagnosed as epileptic. In 317 case histories of manic-depressive psychosis Krisch found that epileptic manifestations were rare. Manic attacks and depression states are rare also in epilepsy; concurrent epilepsy and manic-depressive insanity, extremely rare. A relation between the two diseases is therefore unlikely.

The so-called typical "epileptic character" with his obstinacy, egocentricity, irritability, etc., was by no means common in this group. Except in a third of the patients in whom slowness and dulness appeared to be a common trait, no uniform types were discerned. Classifying 107 cases into personality "types" before the onset of convulsions, sixty-four, a majority, were in normal groups. Moreover, in a mixed group of soldiers, the epileptic men could not be differentiated from the rest by their personality characteristics.

The bibliography is good.

DIE ZERGLIEDERUNG DES PSYCHISCHEN KRANKHEITSBILDES BEI ARTERIOSKLEROSIS-CEREBRI. By S. J. R. de Monchy. Beihefte zur Monatsschrift für Psychiatrie und Neurologie. No. 17. Pp. 84. Paper. Price, 30 marks. Berlin: S. Karger, 1922.

This is a statistical study of mental reaction-types observed in sixty-two clear-cut cases received at the neuropsychiatric clinic in Amsterdam between 1910 and 1920. Condensed histories of the patients are given at the end of the book, and it is noteworthy that, in many, direct mental examination was not possible, so that the analysis is often based on accounts given by relatives. The material includes three classes of cases: cerebral arteriosclerosis, thrombosis and hemorrhage. In the statistical work, when doubt existed as to the proper classification of a case in the following scheme, the author has followed the practice of assigning one half to one group and one half to the other. Two main groups are formed: (1) six and one half without psychic symptoms, and (2) fifty-five and one half with psychic symptoms. The second

group is then divided according to the type of mental picture into (*a*) cases showing exclusively signs of brain damage (irritable weakness, dementia, delirium, etc.); (*b*) depression; (*c*) manic features; (*d*) psychopathic traits; (*e*) paranoid trends. With each group and type contingent correlations have then been determined by use of the formula of Stern: (1) between the particular type of picture and the individual and familial "Anlage" of like kind; (2) between auditory or visual hallucinations and alcoholism, defective hearing or vision, and psychopathic "Anlage"; (3) between precordial anxiety and the existence of cardiac disease or aneurysm of the ascending aorta, and (4) between the occurrence of epileptiform seizures and a specific epileptic tare in the make-up.

The value of the statistical findings is somewhat diminished by the small number of cases in many of the groups, but the work has evidently been done with care. The results support the view that the form of psychosis bears close correlation with the personality of the one affected by the disease. It is perhaps a little surprising to find no mention of schizophrenic pictures, since the figures show no correlation between the diseases grouped as arteriosclerosis and particular types of personal make-up.

The introductory chapters offer interesting discussions of the variety of factors, endogenous and exogenous, which must be considered, and the importance of which can only be evaluated by statistical methods. The precise definitions of the mode of action, pathogenic, pathoplastic, predisposing or precipitating, of various etiologic moments, are especially good, and will do much to help in the standardization of investigations of this kind.

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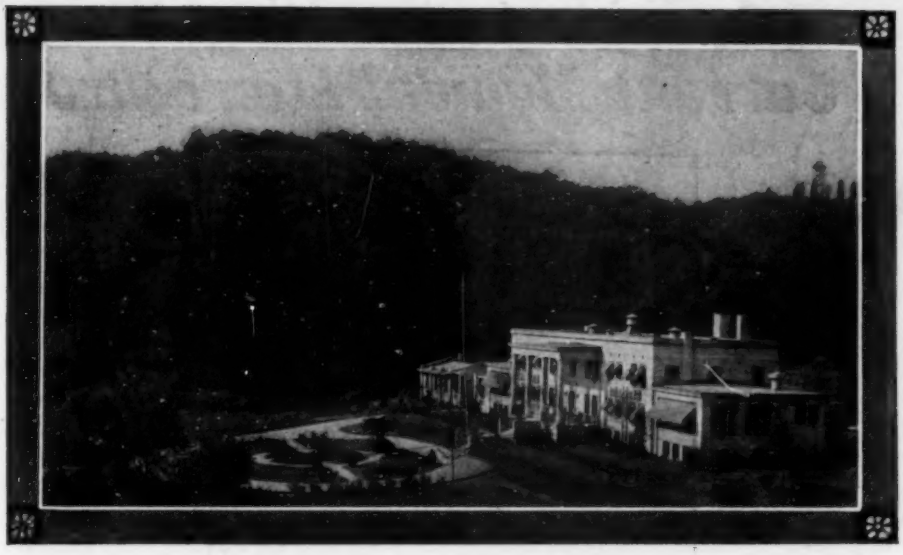

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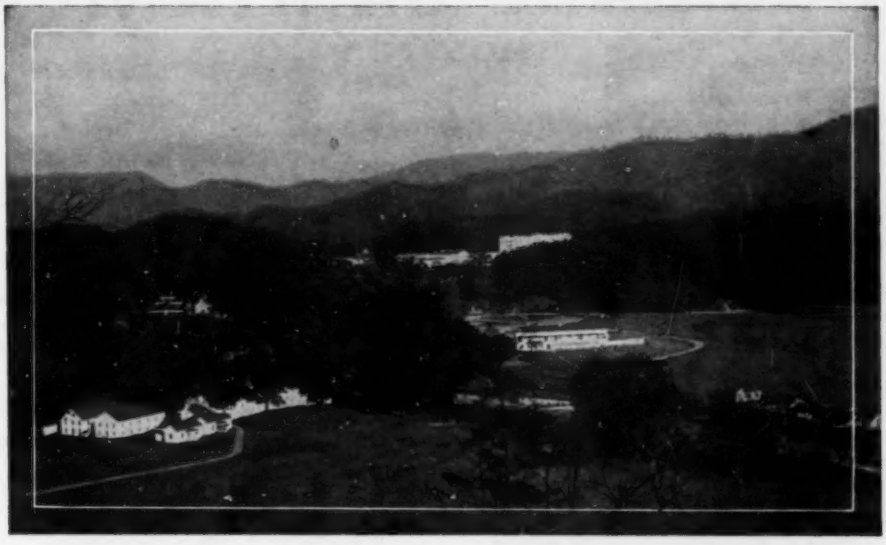

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